A STUDY ON MORTALITY OUTCOMES IN ICU PATIENTS WITH SEQUENTIAL ORGAN FAILURE ASSESSMENT (SOFA) SCORE

Dissertation submitted

in Partial Fulfillment of regulation for the award of

M.D. Degree in General Medicine

The Tamilnadu DR.M.G.R. Medical University

Chennai , April 2015

CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. Santhakumar R.P.S.P** and submitted in partial fulfilment of the requirements for the Degree of **M.D General Medicine**, Branch I of **The Tamilnadu DR. M.G.R. Medical University Chennai.**

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DECLARATION

I solemnly declare that the dissertation titled **"A Study on Mortality Outcomes in ICU Patients with Sequential Organ Failure Assessment (SOFA) Score"** was done by me from AUGUST 2013 to JUNE 2014 under the guidance and supervision of Professor **Dr.KUMAR NATARAJAN .M.D.,**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place : Coimbatore **Dr. SANTHAKUMAR R.P.S.P**

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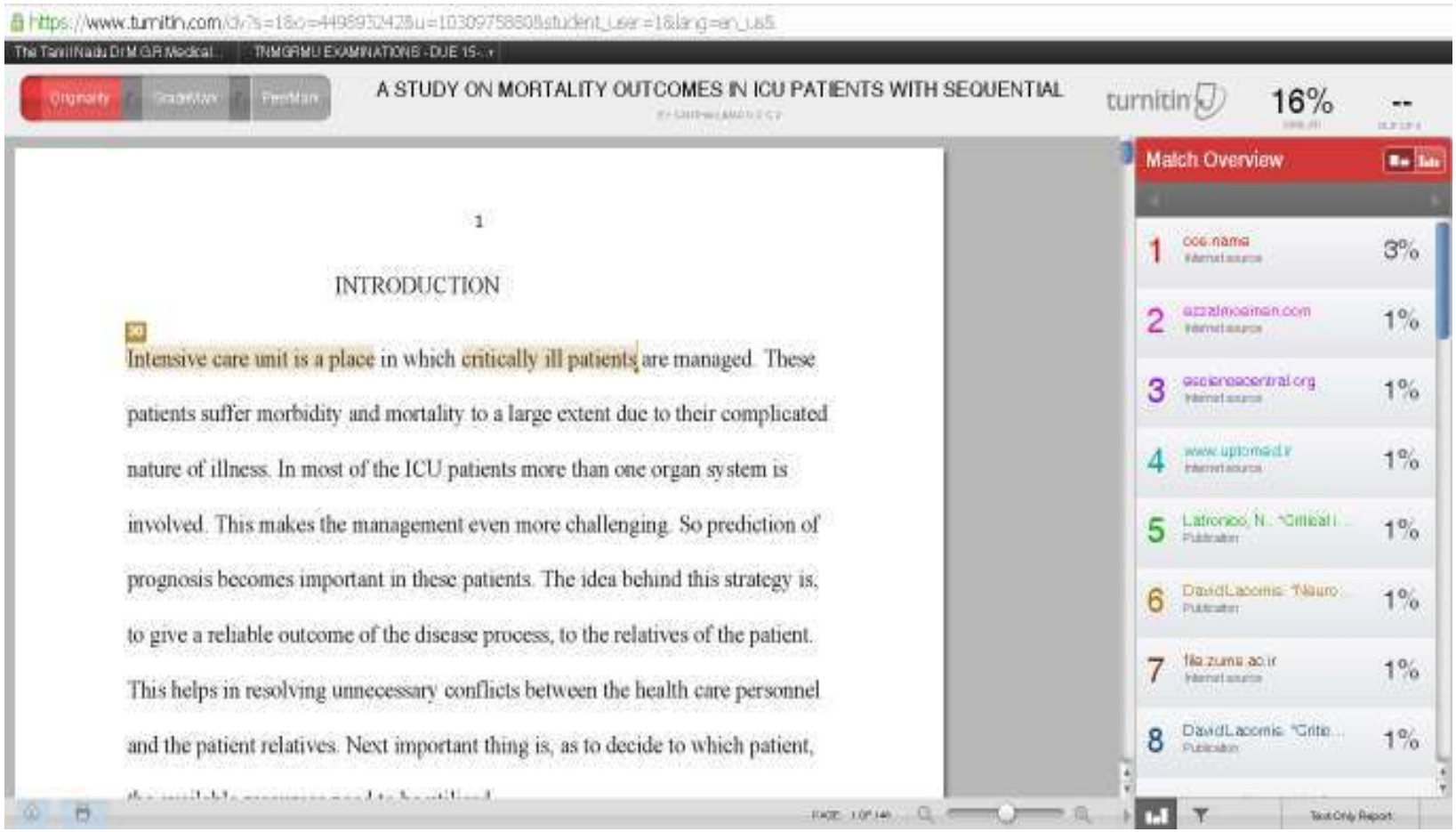
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INTRODUCTION
Intensive care unit is a place in which critically ill patients are managed. These patients suffer morbidity and mortality to a large extent due to their complicated nature of illness. In most of the ICU patients more than one organ system is involved. This makes the management even more challenging. So prediction of prognosis becomes important in these patients. The idea behind this strategy is, to give a reliable outcome of the disease process, to the relatives of the patient. This helps in resolving unnecessary conflicts between the health care personnel and the patient relatives. Next important thing is, as to decide to which patient,
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CONTENTS

ABSTRACT

Purpose of the Project:

This study intends to evaluate the usefulness of sequential organ failure assessment score (SOFA) in assessing organ dysfunction and risk of mortality in patients admitted to ICU.

Background:

Outcome prediction is important in both clinical and administrative ICU management. It can be usefully applied to monitor the progress of an individual ICU. It also provides useful information on likely patient outcomes for critically ill patients and also for therapeutic decision making and using available resources efficiently. In an ICU setting serial organ function monitoring is important since there is a time to time variation in the general condition of the patient. Sequential Organ Failure Assessment (SOFA) is one such outcome prediction model to assess prognosis and mortality risk in ICU patients.

Data Collection and the Source:

All adult patients admitted to the intensive care unit of Coimbatore Medical College Hospital will be included in the study. Blood samples will be collected from the patients admitted to the ICU for the investigations as per the data needed by the SOFA scoring system. Along with these, blood pressure monitoring and Glasgow coma scale evaluation will be done on admission and then for every 48 hrs, for six days or till the patient leaves the ICU either as survivor or non survivor, whichever occurs earlier.

Sampling method:

Prospective observational cohort study.

Case definition:

Patients admitted to the ICU with suspected multi organ failure.

Results:

The age group in this study ranges from 17 to 85. The study shows that above the SOFA score value of 12, there is a sharp increase in mortality rate. More than 85% of the non survivors had an increase in their SOFA scores during their stay in ICU. Admission SOFA and mean sofa are excellent predictors of mortality. Total SOFA provides information about severity of organ failure. Mechanically ventilated patients had a higher mortality rate compared to non ventilated patients. Presence of diabetes and hypertension did not show any significant association with mortality in our study.

Conclusion:

A rise in SOFA score is a strong predictor of mortality in ICU patients.

Key words: SOFA – sequential organ failure assessment. ICU – Intensive care unit.

INTRODUCTION

Intensive care unit is a place in which critically ill patients are managed. These patients suffer morbidity and mortality to a large extent due to their complicated nature of illness. In most of the ICU patients more than one organ system is involved. This makes the management even more challenging. So prediction of prognosis becomes important in these patients. The idea behind this strategy is, to give a reliable outcome of the disease process, to the relatives of the patient. This helps in resolving unnecessary conflicts between the health care personnel and the patient relatives. Next important thing is, as to decide to which patient, the available resources need to be utilised.

This led to the idea of devising scoring systems. These systems guide the efficient utilisation of ICU resources, especially in a resource starved setting. This helps in preventing dumping of valuable drugs and treatment modalities in a patient, who may not survive in spite of all efforts. On the contrary they can be utilised for a person, who may improve well with such costly intervention.

Sequential organ failure assessment called the SOFA scoring system is a simple scoring system calculated using easily available basic investigations, to predict outcome, especially mortality in ICU patients.

This study was undertaken to evaluate the score among ICU patients in Coimbatore medical college hospital admitted with various systemic illness and features of multi organ dysfunction.

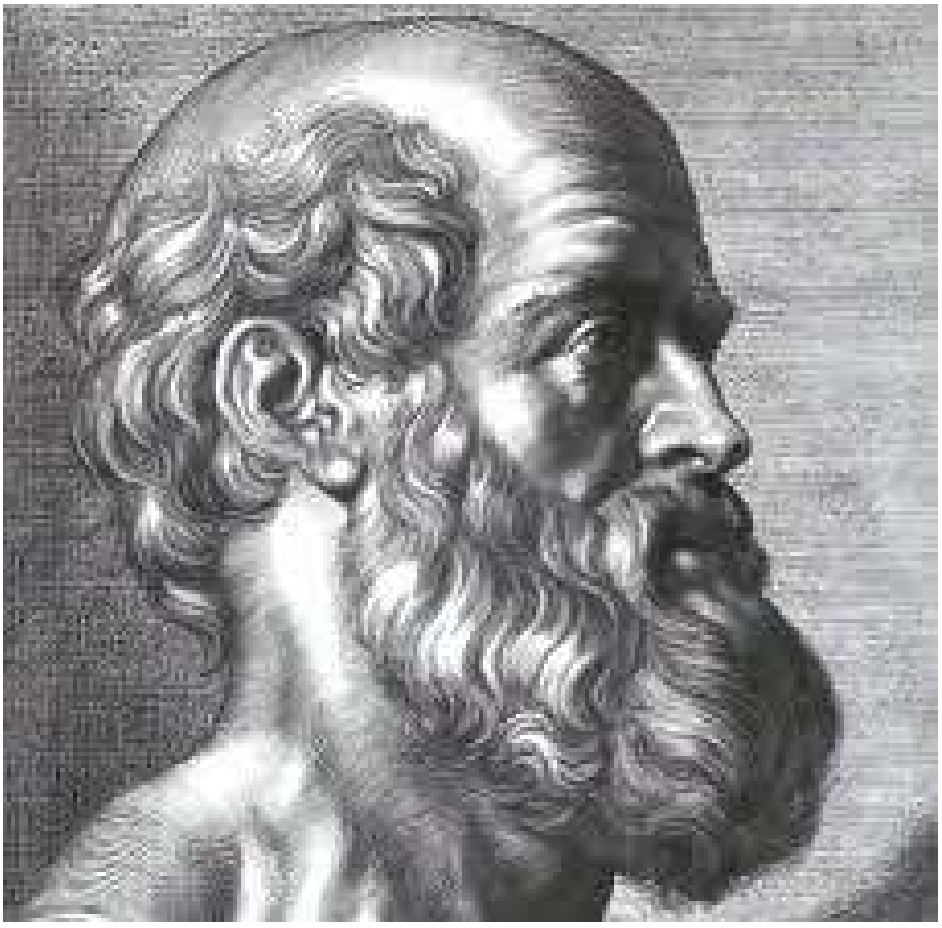
AIMS OF THE STUDY

- To study the usefulness of sequential organ failure assessment score in predicting mortality among ICU patients.
- To study the impact of comorbid illnesses like diabetes and hypertension on outcome, in ICU patients in relation to SOFA score.
- To study the mortality among mechanically ventilated patients and its correlation with SOFA score.

REVIEW OF LITERATURE

I would especially commend the physician who, in acute diseases, by which the bulk of mankind are cut off, conducts the treatment better than others.

- Hippocrates



ICU and Critical care:

The management of critically ill patients admitted to the intensive care units are becoming more and more challenging due to emergence of new diseases day by day, the combination of various illness and the increased life expectancy of population due to advanced medical care. New treatment protocols are being formulated and older treatment protocols are revised periodically for patient management and care in an ICU setting, from the previous experiences and with the help of newer drugs coming up every day. An ICU patient is totally different from a patient in the general ward, right from his physiology, disease pathogenesis, nutritional requirements, response to treatment and overall prognosis. This indeed makes them a population which needs special care in all aspects. Most of the ICU patients have more than one disease process which is manifested by the vital organ involvement. This gave rise to the concept of multiorgan dysfunction syndrome(MODS), which means the abnormal function or failure of more than one organ system.

So the standard of care for these set of population lies in proper diagnosis, monitoring of treatment response and progress every day and even every hour to ensure proper outcome, which means survival from that illness or atleast not to succumb to the illness. To ensure this we need to understand

the nature of illness a critical care unit patient is going through, the pathogenesis of the disease process and its prognosis.

In addition to their inherent disease process, a critical care unit patient is also more prone for nosocomial infections due to improper nutrition, immunodeficient states, systemic illness like diabetes, hypertension etc. Geriatric patients falls under an even more riskier group. So all these factors add fuel to the entity called multi organ dysfunction syndrome(MODS).

Multi organ dysfunction syndrome:

The abnormal function or failure of more than one organ or organ system requiring medical support to maintain homeostasis is called MODS. In a susceptible individual, under the influence of associated comorbidities, the organ systems fail one by one ultimately leading to a complicated disease process and death.

Pathogenesis:

The general principles governing the syndrome of multiorgan dysfunction are,

1) Organ failure, no matter how defined, must persist beyond 24 hours
2) Mortality risk increases as the patients accrue additional failing

3) Prognosis is worsened by increased duration of organ failure.

These observations remain true across various critical care settings all over the world. Systemic inflammatory response syndrome (SIRS) is the common basis for multi organ system failure. Infection is by far the commonest cause of SIRS. Though other triggers like pancreatitis, trauma and burns etc can also elicit a similar response.

Systemic inflammatory response syndrome:

It includes more than two of the following:

1) Rise in temperature >38 degree celcius or hypothermia (<36 degree celcius)
2) Tachypnoea (respiratory rate>24 /min)
3) Heart rate > 90/min
4) Leukocytosis (>12 $\times 10^3$/microlitre), leukopenia (<4 $\times$ 10^3/microlitre).

Pathophysiology:

Bacteria and fungi trigger most cases of sepsis which are less or not harmful to immune competent host. These organisms exploit the host defence mechanism to establish life threatening infections. Once these organisms enter the blood , the body can build up a vigorous immune response, that results in severe sepsis but not able to kill the organism. This results in what called systemic inflammatory response syndrome.

An ICU patient:

A slender and restricted diet is always dangerous in chronic and in acute diseases.

- Hippocrates 400 BC

A lot of changes are produced in the metabolic milieu of a patient admitted in critical care. They are

- Poor food intake
- Prolonged bed rest
- Changes in substrate utilization
- Stress due to illness or surgical procedure
- Hypermetabolism
- Exogenous steroids
- Immobility

All these leads to a process termed as 'autocannibalism', which indicates the loss of lean and fat body mass. This leads to a state of malnutrition.

Malnutrition can lead to the following consequences

- Increased morbidity and mortality
- Prolonged hospital stay

- Reduced respiratory and cardiac function
- Impaired wound healing
- Increased risk of infection due to immunosupression.

A lot of guidelines are available on how to give nutritional support in an ICU patient. Some of them are

1) National institute for health and clinical excellence(NICE): Nutritional support in adults (2006).
2) European society for parenteral and enteral nutrition (ESPEN): Enteral nutrition (2006).
3) Intensive care society (ICS) 2005: Practical management of parenteral nutrition in critically ill patients.
4) Canadian critical care network 2003/2007: clinical practice guidelines.

Nutritional requirements:

Basal energy expenditure can be calculated for each individual by the Harris – Benedict equation

For men, BEE = 66.5 + (13.75 × weight in kg) + (5.003 × height in cm) – (6.775 × age in years)

For women, BEE = 655.1 + (9.563 × weight in kg) + (1.850 × height in cm) – (4.676 × age in years).

Requirements per day:

- Energy – 25 to 30 kcal/kg
- Water – 30 ml/kg
- Carbohydrate – 55 to 70 % of total energy
- Fat – 15 to 30 % of total energy
- Protein – 10 to 15 % of total energy

Complications of Enteral nutrition:

1) The feeding tube can get dislodged or get blocked.
2) Gastric hypersecretion
3) Lactose intolerance
4) Hyperosmolar feeding
5) Malabsorption
6) Altered bowel flora

In short, all these dietary factors should be taken into consideration during planning of patient in an ICU.

Some of the commonest causes of ICU admission are

1) Systemic infections
2) Sepsis
3) ARDS
4) Acute coronary syndrome

5) Renal failure
6) Acute neurological illness
7) Cancer and oncological emergencies

Systemic infections:

Infections were once a greatest threat to mankind. With the advent of antibiotics lot of dreadful infections were brought under control. With the evolution of human immune deficiency virus many new infections emerged and previously drug sensitive microbes became resistant. Also in asian countries , india especially higher prevalence of diabetes and poor hygienic conditions added to a higher incidence of infections. So antibiotic resistant microbes are the concern of this century. Some of the commonest infections encountered in the ICU are

1) Urinary tract infections and
2) Pneumonias
3) Meningitis

Urinary tract infections:

The commonest and most challenging infection in any hospitalised patient is UTI. It can range from simple uncomplicated infection to a serious life threatening infection. It can vary in spectrum from asymptomatic bacteruria to cystitis, prostatitis and pyelonephritis.

UTI is common in women of reproductive age group(1,2) and men after the age of 50 due to prostrate hypertrophy. In ICU patients the predisposing factor for UTI are indwelling urinary catheters, renal stones, abnormal micturition and significant residual urine due to inability to void or incomplete voiding which is common in a long term bed ridden patient.(3,4)

Pathogens:

The organisms implicated in UTI in the order of prevalence are

1) Escherichia coli
2) Staphylococcus saphrophyticus
3) Klebsiella
4) Proteus
5) Enterococcus
6) Citrobacter
7) Salmonella
8) Candida species and other organisms

Patient presents with or without fever with chills and rigor and elevated blood counts. Prompt diagnosis and management are essential to prevent severe life threatening infections.

Untreated or improperly managed UTI leads to complications like recurrent UTI, chronic pyelonephritis and renal insufficiency(5) either acute or chronic. When the infection overwhelms especially due a drug resistant organism this can lead to systemic sepsis ,also called urosepsis.

Pneumonias:

Patient may be admitted in an ICU for a community acquired pneumonia or the infection can be nosocomial in patients admitted for some other illness. In either form this infection can be life threatening needing mechanical ventilator support.

Ventilator Associated Pneumonias :

ICU patients are more prone to get lung infections especially pneumonia. Ventilator associated pneumonia(VAP) is common in patients requiring mechanical ventilation. In any given day at least 10% of patients will have pneumonia in an ICU. Factors responsible for VAP are

1) Colonisation of oropharynx with pathogenic organism
2) Aspiration of these orgasnisms into lower respiratory tract
3) Impaired host defence mechanism

Rupture of distal airspaces during mechanical ventilation due to overdistension is called volutrauma and pressure related lung injury is called barotrauma(6). This leads to infiltration of distal airspaces with exudative substances. This condition is called Ventilator Induced Lung Injury(VILI)(7). The cytokines produced in the lung could enter the systemic circulation and produce widespread inflammatory injury in the distant organ and cause multiorgan failure(8)Morbidity and mortality is more common with this condition. But patients when given adequate care in a high intensive ICU, mortality can be reduced(9) .

Meningitis:

Types of meningitis:-

Meningitis can be categorized according to CSF cytochemical picture as, neutrophilic meningitis, lymphocytic meningitis, and aseptic meningitis.

Neutrophilic meningitis:

The most common cause is bacterial infection. Other rare causes are fungal infection, nocardian infection, actinomyces infection etc.

Bacterial meningitis:

Acute bacterial infection of meninges causes a clinical picture of acute meningitis. Also known as bacterial meningitis, purulent meningitis and septic meningitis. Most often meninges , subarachnoid space and brain parenchyma are all involved in inflammatory reaction causing meningoencephalitis.

Currently most common organisms responsible for community acquired bacterial meningitis are streptococcus pneumonia, meningococcus, Haemophilus influenza, Group *B* Streptococcus and Listeria monocytogenes. Neisseria meningitidis and Streptococcus

pneumoniae are the most common pathogens in patients without immune deficiency.

Streptococcus pneumoniae, is the most common cause of meningitis in adults above 20 years. The predisposing conditions are pneumonia due to pneumococcus, sinusitis, mastoiditis, otitismedia, choclear implants, diabetes mellitus, postsplenectomy.

Neisseria meningitides is most common in adolescent and young adults(2- 20 years). The bacteria are usually recovered from blood or cutaneous lesions before meningitis starts, indicating that spread to the CNS is hematogenous. Occasionally it may gain access directly from the nasopharynx through cribriform plate. Found in individuals with complement deficiency.

In infants and children most common organisms are Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenza type b.

In infancy most common pathogens are Group B Streptococcus, Escherichia coli, Listeria monocytogenes is frequently reported in meningitis above 50 years, especially adults with chronic diseases (e.g. patients on haemodialysis, malignancy, connective tissue disorders).

Staphylococci (S. aureus and S. epidermidis) and gram-negative bacilli are common pathogens in patients following a neurosurgical

procedure. Sometimes it is a complication of cavernous sinus thrombosis, subdural or epidural abscess.

Recurrent Bacterial Meningitis signal a host defect, either in local anatomy or in antibacterial and immunologic defenses.

1. **Local anatomy:**

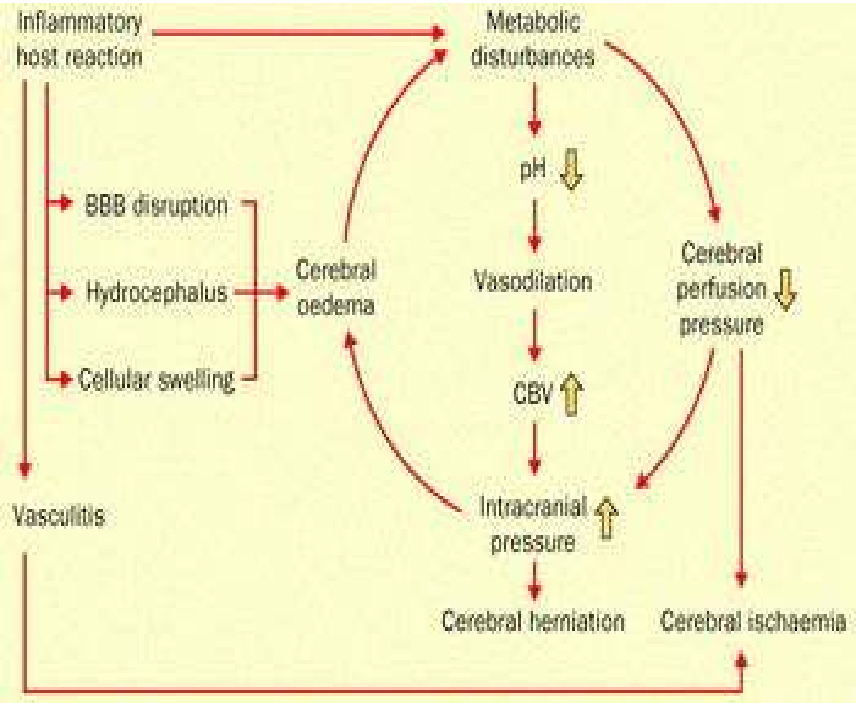
- Head trauma (floor of the anterior cranial fossa) with CSF rhinorrhoea
- Head trauma(temporal bone)with access of bacteria to the CSF from the ear
- In situ shunt devices for relief of hydrocephalus.

2. General (defective immunologic defenses):

- Diabetes mellitus
- immune deficiency or immunosuppression.

A pathogenesis of bacterial meningitis is inflammatory reaction induced by invading bacteria. Most often due elevated CSF cytokines and chemokines. The inflammatory reaction is severe in the subarachnoid space over the brain and around the cisterns (base of the brain). It may extend along the perivascular spaces into the brain and spinal cord but rarely breaks into the parenchyma of the brain.

Pathogenesis of meningitis



The classical clinical triad of meningitis consists of fever, headache, and neck rigidity. Nausea, vomiting, phonophobia, photophobia are usually seen. With the disease progression, the sensorium becomes clouded and stupor. Convulsive seizures are often an early symptom (20%-40% cases), especially in children.

The temperature is elevated at 101°F to 103°F. The pulse is usually rapid and there is increased respiratory rate. There is nuchal rigidity, Kernig's sign (resistance to extension of the legs) and Brudzinski's signs (resistance to forward flexion of the neck). The above signs may be absent in newborn and elderly. Tendon reflexes are often decreased.

Cranial nerve palsies and focal neurologic deficits are uncommon and usually develop several days after the onset of infection. Papilloedema may develop if the meningitis persists for more than a week otherwise optic disc is normal.

The WBC count is increased and it is usually in the range of 10,000/mm^3 to 30,000/mm^3. The pressure of CSF is increased and usually between 200 and 500 mm H_2O. The CSF is cloudy because it contains a numerous cells, predominantly polymorphonuclear leukocytes. The cell count in the CSF is usually between 2,000/mm^3 and 10,000/mm^3. The protein content of CSF is increased. The sugar content is decreased (below 20 mg/dl). Particle agglutination testing may rapidly identify bacterial antigens in the CSF.

Another modality used for the evaluation for bacterial meningitis is polymerase chain reaction (PCR.) of the CSF. PCR has high sensitivity and specificity for the detection of bacteria such as S. pneumonia in the CSF, but false-positive results have been reported. Other tests include real time PCR for rapid diagnosis of bacterial meningitis. Fluorescence In situ Hybridization (FISH). PCR is highly sensitive tool for rapid diagnosis of bacterial meningitis but it is costly. FISH is useful for the identification of CSF samples which shows multiple bacteria during gram staining.

Management in ICU:

- Fluid management
- Antibiotic therapy
- Anti oedema measures for cerebral oedema
- Anti epileptics if patient is having seizures
- Physiotherapy
- Ventilator support if patient is in respiratory arrest

ICU sepsis:

Sepsis is more dangerous in patients in critical care.

Severe sepsis is defined as one or more of the following

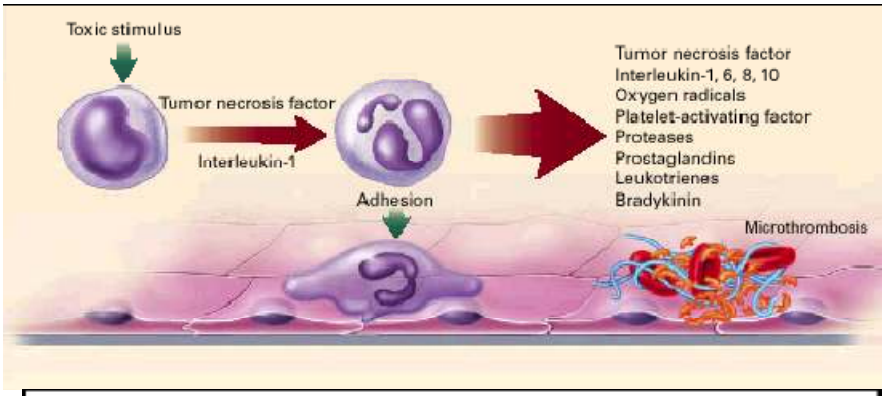
1. Blood pressure $\leq$ 90 mm of Hg or mean arterial blood pressure $\leq$ 70 mm of Hg that responds to administration of intra venous fluid.
2. Urine output <0.5ml/kg/hr for 1 hour despite adequate fluid resuscitation.
3. PaO2/FiO2 $\leq$ 250 or if lung is the only dysfunctional organ PaO2/FiO2 $\leq$200
4. Platelet cant < 80,000/ microlitre or 50 percent decrease in platelet over previous 3 days.
5. Unexplained metabolic acidosis P_H<7.3 or a base deficit $\geq$ 5 meq/litre and a plasma lactate level of >1.5 times upper limit of normal

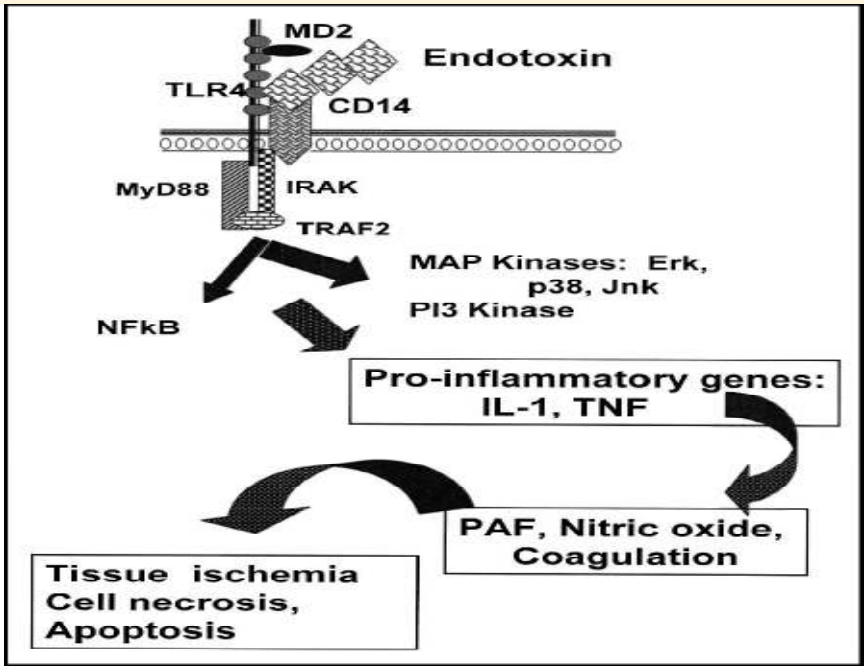
6. Pulmonary artery Wedge pressure ≥ 12 mm of Hg or central venous pressure ≥ 8 mm of Hg.

Septic shock is systolic blood pressure <90mm of Hg for at least 1 hour despite adequate fluid resusitation or need for vasopressors to maintain systolic blood pressure ≥ 90 mm of Hg or mean arterial pressure ≥70 mm of Hg.

Pathogenesis and complications:

Pathogenesis:





Complications:

1. **Cardio pulmonary complications:**
   - Can be due to ventilation- perfusion mismatch and increasing alveolar epithelial injury – Acute respiratory distress syndrome and sepsis induced hypotension due to decreased effective intravascular volume.
2. **Critical illness related corticosteroid insufficiency** – manifesting as refractory hypotension.
3. **Renal failure:**

Pre renal and acute renal failure due to acute tubular necrosis are common.

4. **Coagulopathy:**

Thrombocytopenia and disseminated intravascular coagulation are common.

5. **Neurologic complications:**
   - Weaning from ventilator becomes difficult in the presence of critical care polyneuropathy.
6. **Immunosupression:**

– Patients with severe sepsis are profoundly Immuno suppressed (10)

Management :

1) Hemodynamic resuscitation and acute life support
2) Infection control
3) Organ support and minimizing health care associated injury
4) Interventions to modify host inflammatory response

These are the four important principles of sepsis management in ICU (11).

Adult Respiratory Distress Syndrome:

ARDS is the most common non infectious cause of lung infiltrates in ICU (12). This terminology was coined in the year 1967. It is otherwise called “shock lung”. Multi lobar pneumonia may be difficult to distinguish from an ARDS.

Some of the commonest causes of ARDS are (13)

1) Gram negative sepsis
2) Pneumonia
3) Near drowning
4) Toxic fumes inhalation
5) Multiple blood transfusions
6) Aspiration of gastric contents
7) Burns
8) Pancreatitis

Pneumonia patients are at 10% risk of developing ARDS. In contrast 50% of those with ARDS develop pneumonia during their ICU stay(14).

Symptoms:

Dyspnoea , tachypnoea and hypoxemia(15,16)

Diagnostic criteria:

BERLIN CRITERIA 2012 – EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE(17,18):

1. Acute onset lung injury less than 1 week of an obvious clinical insult and with progression of respiratory symptoms
2. Opacities on both lung fields not explained by other pulmonary pathology (e.g. pleural effusion, pneumothorax, or nodules)
3. Respiratory failure not due to heart failure or volume overload
4. Fall in arterial PaO2/FiO2 ratio:

   Mild : 201 - 300 mmHg (≤ 39.9 kPa)

   Moderate: 101 - 200 mmHg (≤ 26.6 kPa)

   Severe: ≤ 100 mmHg (≤ 13.3 kPa)

Principles of ARDS management in ICU:

1) Low volume ventilation(19)
2) Permissive hypercapnia(20)
3) Promoting oxygen transport by improving cardiac output and haemoglobin levels by transfusion, if needed(21 - 23)
4) Low PEEP (positive end expiratory pressure)(24)
5) Steroids in the fibrinoproliferative phase(25) which promotes collagen breakdown and inhibits fibrosis(26)

Multiorgan failure is the cause of death in majority of the ARDS cases. Less than 40% deaths are due to respiratory failure(27 - 31). This highlights the importance of integrated management in ARDS. So the management strategy in an ICU for ARDS should focus not on the lungs alone.

Acute coronary syndromes:

Cardiac illness takes away the major toll of human life. Coronary artery disease in 2004 represented 29% of global deaths killing 17.1 million people worldwide according to WHO estimates.

Acute myocardial infarction occurs when the coronary perfusion is not able to meet myocardial contractile demand (32). In an ICU setting MI is often underreported due to inability to communicate under

influence of sedation, analgesic drugs, trauma and sepsis. MI in ICU is associated with higher morbidity, mortality, complications and healthcare cost(33,34).

In 2012, the Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Health Federation (ESC/ACCF/AHA/WHF) redefined MI as a rise and/or fall of cardiac biomarkers with at least 1 value above the 99th percentile of the upper reference limit together with evidence of myocardial ischemia with at least 1 of the following (35).

1. symptoms suggesting myocardial ischemia
2. Q waves on electrocardiogram (pathological)
3. New onset ST-T changes or left bundle branch block (LBBB)
4. Acute loss of viable myocardium or a new regional wall motion abnormality
5. Diagnosis of intracoronary thrombus by angiography or autopsy
6. Sudden, unexpected cardiac death with symptoms suggestive of myocardial ischemia and presumed new ST-segment elevation
7. LBBB, and/or evidence of fresh thrombus by coronary angiography and/or autopsy.

So, Clinical signs and symptoms, electrocardiography, cardiac enzymes are vital in the diagnosis of acute MI(36).

Management of acute MI in ICU(37 - 44):

The scenario of acute MI management differs for critically ill and non critically ill patients. Comorbidties like renal failure, coagulopathies, sepsis, mechanical ventilation etc complicate the management.

The principle treatment modalities are

1) Oxygen, to maintain Sao2 > 90%
2) Fibrinolysis, with streptokinase or r-tPA
3) Nitrates for pain relief
4) Morphine to relieve excruciating pain
5) Anticoagulants and statins
6) Antiarrhythmics
7) Beta blockers
8) Antiplatelets

Percutaneous coronary intervention:

In spite of all these non invasive strategies of management, early PCI offers a very favourable outcome in acute coronary syndrome, in an ICU patient (45 - 48). However coronary angiography is associated with adverse outcomes in critically ill patients(49).

Poisoning:

All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy."

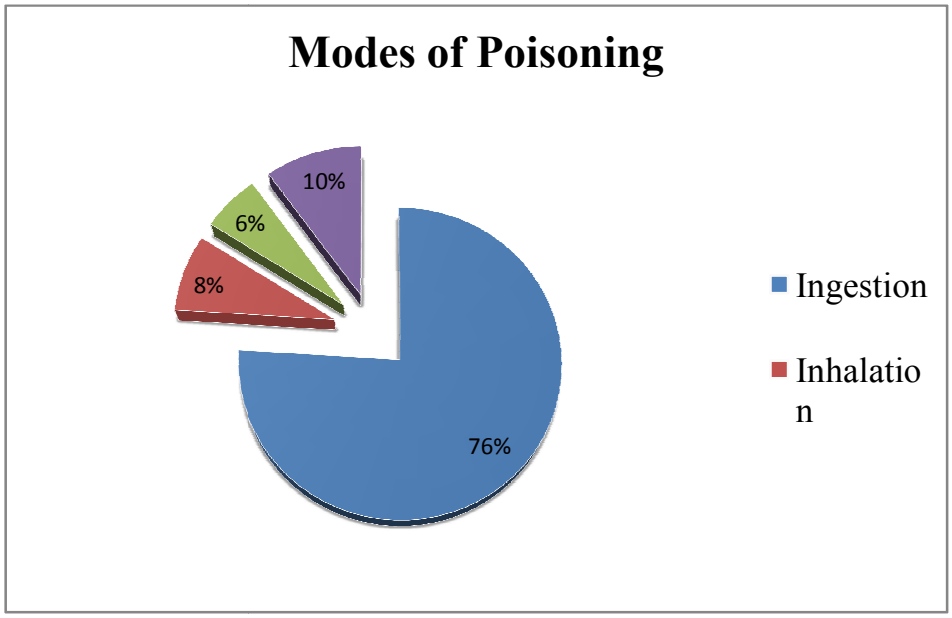
- Paracelsus

Poisoning cases utilise a significant amount of ICU resources. It is also one among the commonest cause of emergency ICU admissions(50,51). These patients also pose an immense diagnostic as well as therapeutic challenge because of the varied toxicology profile in each individual according to the agents they consumed. That depends on the socioeconomic status and the availability of agents at a particular point of time. The outcome of these patients depend on dose of the agent consumed, comorbid illness, time from consumption to arrival at the treatment centre and quality of the health care provider(52 - 56).

In India, the commonly consumed poison is agricultural pesticides(57), followed by alcohol intoxication, drug overdose. Incidence of poisoning is more among males and it has been noted in various studies across India (58,59).

Modes of poisoning:

Majority of the poisoning occurs via ingestion of the substance. Most of them are suicidal and a few are accidental poisoning. The trend is depicted in the following pie chart



Management in ICU:

Assessment of clinical features:

Some of the manifestations upon which one should suspect an intoxication are (60)

1) History of drug overdose
2) Prior suicidal attempts
3) History of psychiatric illness
4) Delirium
5) Rotatory nystagmus

6) Cardiorespiratory arrest
7) Arrhythmias
8) Multiorgan failure
9) Rhabdomyolysis
10) Metabolic acidosis or alkalosis

Toxicology screening:

Some of the commonly abused drugs detected by urine screening are(61)

1) Amphetamines
2) Barbiturates
3) Benzodiazepines
4) Cannabinoids
5) Cocaine
6) Phencyclidine
7) Opioids.

Treatment:

Patients should be assessed for their level of consciousness. Any head injuries or spinal cord injuries should be ruled out. Some of the management strategies used in ICU are

- Coma cocktail, comprising of thiamine, dextrose and naloxone can be given. It can be both diagnostic and therapeutic(62).
- Flumazenil can be given if benzodiazepine overdose is suspected (63)
- Apart from this, removal of toxin exposure can be done by skin decontamination, ocular and gastric decontamination.
- Toxins absorbed through GI tract can be cleared by

  1) Emesis
  2) Gastric lavage
  3) Whole bowel irrigation
  4) Activated charcoal with cathartic
- Urinary alkalization for compounds like salicylic acid, Phenobarbital, methotrexate, isoniazid etc.
- Hemoperfusion
- Hemodialysis for renal failure due to nephrotoxic compounds
- Antidotes can be used for specific poisons. One of the importance of identifying the compound taken is for the purpose of administering antidote. Some of the commonly used antidotes in ICU are

Acetaminophen	**N acetyl cysteine**
Anticholinergic	**Physostigmine**
Anticholinesterase	**Atropine**
Cyanide	**Amyl nitrite, sodium nitrite**
Digoxin	**Digoxin specific antibody**
Methanol	**Ethanol/ fomepizole**
Opioids	**Naloxone**
Organophosphate	**Atropine**

Corrosive substances:

Corrosive agents are present in many house hold things we use daily. It is available in various forms and it is one among the commonest substances ingested or got exposed to either intentionally as in case of suicide attempt, accidentally as in a workplace.

Most of the agents contain either acid or an alkali. The pathophysiology of injury caused by both acid and alkali are different.

Alkali:

The mechanism of damage due to ingestion of alkali is by formation of hydroxyl ions due to contact of alkali with the tissue. It causes liquefaction necrosis. Collagen destruction, fat saponification,

emulsification of cell membrane, transmural thrombosis and cell death. All these leads to vascular thrombosis in the area of contact.

Acid:

Acid injury causes coagulation necrosis. Contact of acid with tissue surface causes cellular protein dessication, denaturation and precipitation.

Alkali ingestion causes severe damage to oesophagus followed by stomach due to increased tissue binding of alkalis and acid ingestion causes more injury to stomach, sparing the oesophagus most of the times, due to rapid transit of acid down the oesophagus.

Concerns in ICU:

Corrosive ingestion, when the manifestations are severe it can cause metabolic acidosis, disseminated intravascular coagulation and which in turn can lead to a multiorgan dysfunction.

Management:

First hand management comprises of securing the airway which may be injured or stenosed due to the corrosive substance. Airway should be secured using a an endotracheal tube using fibreoptic endoscopy, to avoid injury to edematous tissue causing perforation, by the method of blindly poking an endotracheal tube as which is used routinely.

An ET tube of smaller size at least 1 – 2 cm than which is routinely used, should be used to secure the airway in the presence of laryngeal edema.

Parenteral steroids relieve airway edema to some extent.

At times patient may need emergency procedures like tracheostomy in the presence of severe airway edema, in which the insertion of an endotracheal tube may be difficult.

Milder forms of injury settles with medical management. While severe forms of injury may need surgical intervention.

Snake bite:

Most of the Indian population lives in rural areas where agriculture is the predominant occupation. Snake bite is a common health hazard in these areas. In 1953 Porge pointed out that over 25,000 human lives were taken away every year due to poisonous snake bite in the Indian subcontinent (64)

Government of India data, cited by WHO , 2010 stated that 50,000 deaths have been reported each year(65).

Nearly 216 species of snakes are identified in India, of which 52 species are poisonous.

Medically important species of snakes are cobra, common krait, russell's viper and phursa.

Among these elapidae and viperidae are of highest medical importance and are classified as category I, which results in high levels of morbidity, disability and mortality.

Snake venom:

Ophitoxaemia is the term used to describe the clinical spectrum of snake bite envenomation.

Snake venom is a modified saliva is produced by special glands of snakes. Zootoxin is secreted by glands which are a modification of the parotid salivary gland and are situated below and behind the eye on either side, encapsulated in the muscular sheath.

Large alveoli in the glands store the venom before being conveyed by the duct to the tubular fangs through which it is injected.

It is injected either subcutaneously or intramuscularly.

Chemistry:

Snake venom is a composition of protein, enzymes, cytotoxic substances, neurotoxins, coagulants and anticoagulants.

It has acidic Ph.

Specific gravity is 1.03 and is water soluble.

Digestion of proteins is caused by oxidases and proteases Phosphodiesterase A2 causes hemolysis by lysing RBC membranes

Neurotoxins:

1) Fasciculins
2) Dendrotoxins
3) Alpha neurotoxins

Cytotoxins:

1) Phospholipases
2) Cardiotoxins
3) Haemotoxins

Signs of envenomation:

Systemic manifestations:

Most often the patients complain of

- Abdominal pain
- Nausea
- Vomiting
- Weakness
- Hyperaesthesia of abdominal skin

- Weakness
- Drowsiness
- Ptosis
- Respiratory and bulbar paralysis
- Cardiogenic and vasogenic shock
- Myoglobinuria and renal tubular acidosis with bite of sea snakes

Local manifestations:

- Fang marks can be seen in the bite site
- Cellulitis
- Abscess formation and necrosis
- Pain
- Serosanguinous discharge
- Myalgia
- Regional lymphadenopathy
- Gangrene formation may lead to limb loss

Properties of snake venom according to species and lethal dose:

Species	Venom ejected per bite	Lethal dose
Cobra	**200 mg**	**15 mg**
Russell's viper	**150 mg**	**40 mg**
Krait	**22 mg**	**1 mg**
Echis	**4.6 mg**	**4 mg**

Management of snake bite in ICU:

Aggressive management strategies should be adopted in managing cases. When intervened promptly morbidity and even mortality could be avoided.

A quick and detailed history is important along with physical examination with special attention to the bitten limb.

General examination, cardiovascular, pulmonary and neurological examinations should be done to assess systemic involvement and associated complications.

Investigations:

- Complete blood counts
- Renal function test
- Urine routine examination
- Coagulation profile
- ABG analysis
- Liver function test
- Creatine phosphokinase
- Saturation monitoring

Treatment strategies:

- Tetanus toxoid should be administered to all patients
- Broad spectrum antibiotics should be administered to prevent spread of infection
- Anti snake venom
- Mechanical ventilation if patient is having respiratory distress
- Fasciotomy and limb support if compartment syndrome is present
- Blood transfusion, platelet and fresh frozen plasma transfusion as and when needed to manage complications like bleeding due to DIC or septicaemia

Anti snake venom:

The anti snake venom (ASV) used in India is polyvalent. It is effective against russells viper, common cobra, common krait and saw scaled viper. Monovalent ASV is not used in India.

Criteria for ASV administration:

ASV is costly and anaphylactic reactions may occur following administration. Therefore unnecessary usage should be avoided.

ASV should be administered only if any of the following manifestations occur

- Evidence of coagulopathy – WBCT >20 minutes.
- Evidence of neurotoxicity – ptosis, muscle paralysis, external ophthalmoplegia.
- Persistent severe vomiting or abdominal pain.
- Rapid extension of cellulitis.
- Swelling of more than half of the bitten limb.

Neuromyopathies in critically ill:

Sir William osler first described muscle weakness and atrophy occurring during critical illness and sepsis (66). The prevalence varies in ICU from 20 – 90 %.

Cytokines and free radicals released as a result of SIRS, affects the microcirculation of the central and peripheral nervous system(67). Critical illness polyneuropathy occurs as a rare complication of sepsis and multiorgan failure.

Pathogenesis:

The disturbed humoral and cellular response in SIRS and MODS results in increased capillary permeability and endothelial damage. This results in microcirculatory dysfunction with tissue hypoxia (68). Certain patients need corticosteroids and neuromuscular blocking agents which also adds to the oxidative stress.

Both total and reduced form of glutathione is decreased in muscle. This results in mitochondrial injury and oxidative stress.

The weakness can be due to neuropathy, myopathy or polyneuropathy. The term critical care illness weakness (CIW) encompasses all these entities.

Spectrum of CIW:

1) Myopathic component
2) Neuropathic component
3) Neuromuscular junction component
4) Metabolic component

5) Encephalopathic component

Critical illness neuropathy(CIN):

This entity was first described by Charles Bolton in 1984(69). It occurs in 50 – 70% of those who develop SIRS. Since SIRS is more common in ICU setting, CIN gains importance as an important cause of acute polyneuropathy. It is a distal axonopathy of both motor and sensory axons. It presents with flaccid weakness and loss of tendon reflexes.

Nerve conduction studies will show involvement of phrenic nerves (70). This results in difficulty in weaning from mechanical ventilator.

This prolongs the ICU stay and increases the morbidity. Other causes like Guillain – Barre Syndrome (GBS), Botulism, myasthenic crisis and porphyria are also causes for acute weakness in ICU.

Diagnostic criteria for CIN:

1) The patient is critically ill
2) Limb weakness or difficulty in weaning patient from ventilator after non neuromuscular causes such as heart and lung diseases have been excluded
3) Electrophysiological evidence of axonal motor and sensory polyneuropathy.

4) Absence of decremental response on repetitive nerve stimulation. Other acute axonal polyneuropathies such as porphyria, thiamine deficiency etc should be excluded.

Definite diagnosis of CIP – if criteria 1 – 4 is fulfilled.

Probable diagnosis of CIP – if criteria 1,3 and 4 are fulfilled.

Diagnosis in ICU – acquired weakness is established if only criteria 1 and 2 are fulfilled

Critical illness myopathy(CIM):

This is an acute myopathy of critically ill patients. CIM occurs in at least one third of patients treated for status asthmaticus. Electrophysiological and muscle biopsy evidence of primary myopathy is seen in all of the 22 critically ill patients involved in one prospective study. The clinical features overlap with critical illness polyneuropathy and neuromuscular junction blockade. It involves neck flexors, limbs, diaphragm and facial muscles. This causes difficulty in weaning from ventilator.

Some of the forms of myopathy are

1) Acute quadriplegic myopathy
2) Acute necrotising myopathy of intensive care
3) Thick filament myopathy

4) Acute corticosteroid myopathy
5) Acute hydrocortisone myopathy
6) Acute myopathy in severe asthma
7) Acute cortoicosteroid and pancuronium associated myopathy and
8) Critical care myopathy

Diagnostic criteria for critical illness myopathy:

Major:

1) Sensory nerve action potential amplitudes more than 80 % of the lower limit of normal(LLN) in two or more nerves
2) Needle EMG, with short duration, low amplitude motor unit potentials(MUPs) with early or normal full recruitment, with or without fibrillation potentials.
3) Absence of decremental response on repetitive nerve stimulation.
4) Muscle histopathologic findings of myopathy with myosin loss.

Minor:

1) Compound muscle action potentials(CMAP) less than 80 % LLN in two or more nerves without conduction block.
2) Elevated serum creatine kinase.
3) Demonstration of muscle excitability.

As per definition, patient should have developed weakness after the onset of critical illness.

Definitive diagnosis – patients should have all four major criteria

Probable diagnosis – patients should fulfil three major criteria and one or more minor criteria

Possible diagnosis – major criteria 1 and 3 or 2 and 3, and one or more minor criteria

Nerve conduction study in CIM will show (71)

Low amplitude

Broadened or absent CMAPs

Relatively preserved SNAPs

Predictors of outcome:

Limb and diaphragmatic weakness can persist for weeks and months after an episode of CIW.

In a study conducted by Leitjen in 50 patients undergoing mechanical ventilation, he found that 29 patients developed peripheral neuropathy. Those patients also had the highest ICU mortality.

De Jonghe et al have also found that the requirement of prolonged weaning period can be predicted by the incidence of ICU acquired paresis.

It is also important to identify CIW, since it prolongs the stay of the patient in ICU. This is especially important in a resource limited setting to cut down the cost and manage them efficiently.

How to prevent CIW:

1) Early rehabilitation
2) Early passive mobilisation
3) Electrical muscle stimulation
4) Daily cycle sessions with bedside ergometer

These measures have been found to improve the power of quadriceps muscle.

Patient assessment in ICU:

An ICU is the place where critically ill patients are managed and require continuous monitoring. It takes a lot of resources in the form of cost, manpower and costly equipments. With overflow of cases we need to know which group of patients in an ICU needs utmost priority for utilisation of these costly resources. The priority may also change according to the improvement or deterioration of patients on an everyday

basis. So to assess who needs considerable amount of care at one particular point of time we need certain guidelines based on evidence. Here comes the role of scoring systems. These systems were designed based on simple clinical parameters which can be done on the bed side and basic laboratory parameters which are available in most of the centres.

Scoring systems:

A few of the most commonly used such systems are

1) APACHE

2) SOFA

3) Simplified acute physiology score (SAPS)

4) Mortality probability model (MPM)

5) Therapeutic intervention scoring system (TISS)

6) Logistic organ dysfunction score (LODS)

7) Multiorgan dysfunction score (MODS)

Among these SOFA, MODS and LODS are organ dysfunction scoring system. APACHE, SAPS II and MPM II are general severity scoring systems.

APACHE SCORING SYSTEM:

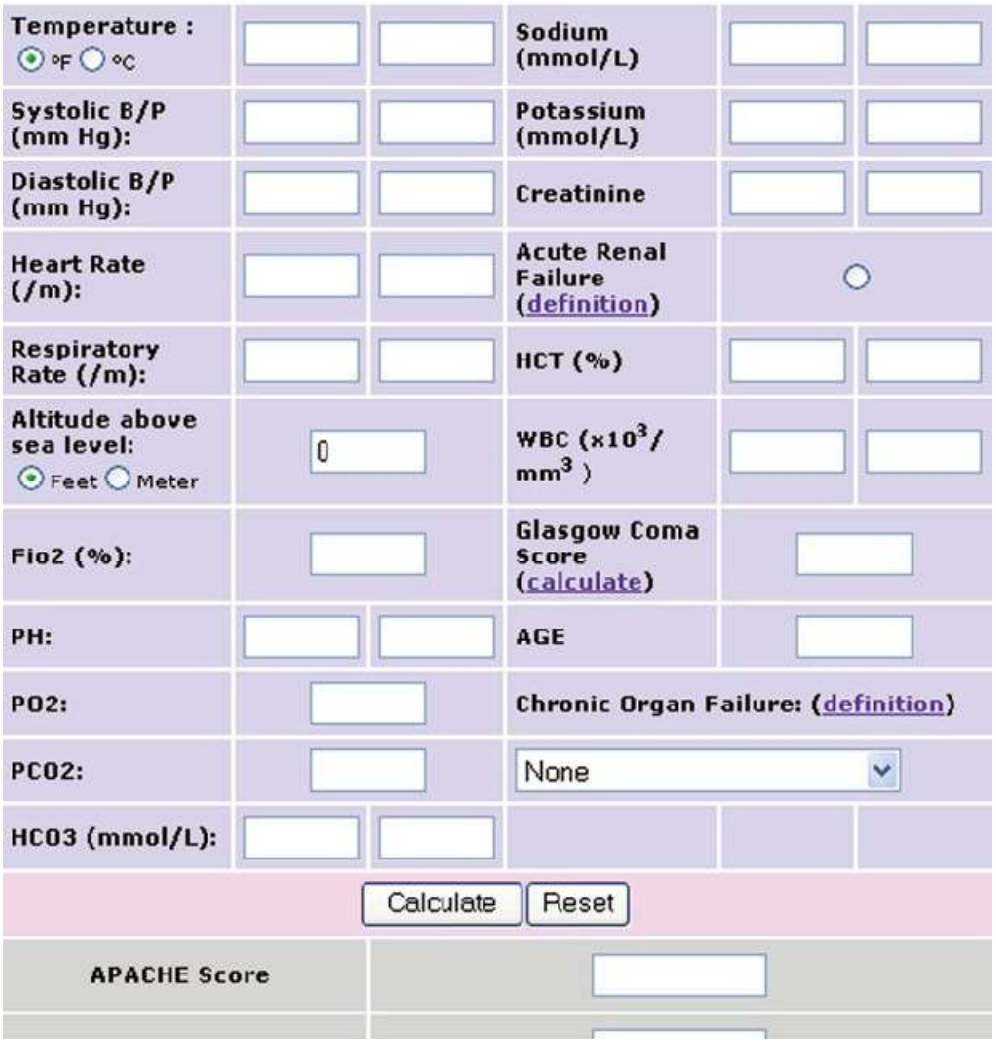
Knaus et al developed APACHE scoring system in 1985(72). It consists of 12 physiological variables calculated by multivariate analysis. The scores ranges from 0 – 71. The data of APACHE II are calculated using the equation

IN HOSPITAL MORTALITY

$$(R/1-R) = -3.517 + (APACHE \square \times 0.146 + S + D)$$

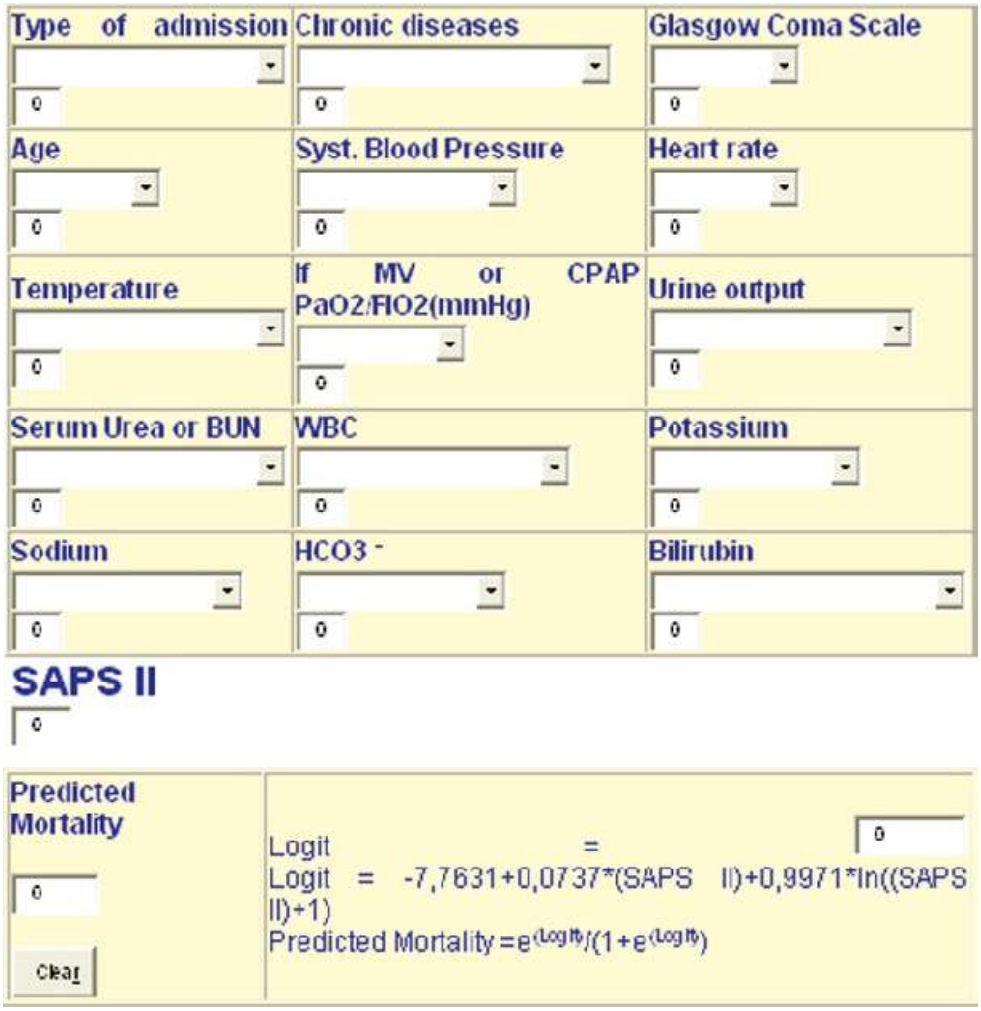
R = Risk of death in hospital, S = Risk due to emergency surgery, and D = Risk due to any specific disease.

A score of 25 or less denotes less than 50% mortality and score of 35 or more denotes more than 85% mortality. Although APACHE II score provides severity of illness of particular group of patients, they provide little information about the risk of individual patients(73). As an improvised version of APACHE II, APACHE III and IV were designed for better prediction.



Simplified acute physiology score(SAPS):

Simplified acute physiology score was introduced by Le Gall et al in 1984(74). It was designed to encounter the difficulties faced during assessment of APS used in APACHE score. It was calculated by taking the 13 most easily measurable physiological variables used in APACHE score. The total score is obtained as the highest score of ICU admission in the first 24 hours. SAPS had its advantage over APACHE II in accurately predicting mortality in a stratified group of patients.



Mortality probability model:

Lemeshow et al in 1985, first published the mortality prediction model(75). He designed 4 models, like probability of death from data collected at ICU admission(MPM_0), Probability of death based on 24 hours data(MPM_{24})probability of death based on 48 hours data(MPM_{48}), probability of death over a period of time based on MPM_0 and change in probability between MPM_0 and MPM_{24} and change in probability between MPM_{24} and MPM_{48}. Lemeshow et al also developed MPM II to assess

serial changes in ICU patients over 72 hours of ICU stay. Hence this model had a better advantage over APACHE and SAPS since these two models lack ability of serial assessment.

(Mortality Probability Models)

Variables (Help)	Values (1 if yes, 0 otherwise)	Beta
Medical or unscheduled surgery admission		0
Metastatic neoplasm		0
Cirrhosis		0
Chronic renal insufficiency		0
C.P.R. prior to admission		0
Coma (Glasgow 3-5) (Help)		0
Heart Rate > = 150		0
Systolic Blood Pressure < = 90 mmHg		0
Acute renal insufficiency		0
Cardiac dysrhythmia		0
Cerebrovascular incident		0
Gastrointestinal bleeding		0
Intracranial mass effect		0
Mechanical ventilation		0
Age	0	0.03057
Predicted Death rate : 0 Compute Clear		Logit = 0 Logit = Sum (values * beta) + age * 0.03057 -5.46836 Predicted death rate = $(e^{Logit}) / (1 + e^{Logit})$

THERAPEUTIC INTERVENTION SCORING SYSTEM:

Cullen et al in 1974 developed this scoring system(76). It utilises 76 monitoring and therapeutic parameters. Scores of the first three day ICU stay correlate well with survival. So it is useful in discriminating survivors and non survivors, according to whether the score increases or decreases, respectively.

(Therapeutic Intervention Scoring System - Update 1983)

4 points		3 points	
a. Cardiac arrest and/or countershock within past 48 h	○yes ○no	a. Central iv hyperalimentation (includes renal, cardiac, hepatic failure fluid)	○yes ○no
b. Controlled ventilation with or without PEEP	○yes ○no	b. Pacemaker on standby	○yes ○no
c. Controlled ventilation with intermittent or continuous muscle relaxants	○yes ○no	c. Chest tubes	○yes ○no
d. Balloon tamponade of varices	○yes ○no	d. IMV or assisted ventilation	○yes ○no
e. Continuous arterial infusion	○yes ○no	e. CPAP	○yes ○no
f. Pulmonary artery catheter	○yes ○no	f. Concentrated K+ infusion via central catheter	○yes ○no
g. Atrial and/or ventricular pacing	○yes ○no	g. Nasotracheal or orotracheal intubation	○yes ○no
h. Hemodialysis in unstable patient	○yes ○no	h. Blind intratracheal suctioning	○yes ○no
i. Peritoneal dialysis	○yes ○no	i. Complex metabolic balance (frequent intake and output)	○yes ○no
j. Induced hypothermia	○yes ○no	j. Multiple ABG, bleeding, and/or STAT studies (> 4 shift)	○yes ○no
k. Pressure-activated blood infusion	○yes ○no	k. Frequent infusion of blood products (>5 units /24 h)	○yes ○no
l. G-suit	○yes ○no	l. Bolus iv medication (nonscheduled)	○yes ○no
m. Intracranial pressure monitoring	○yes ○no	m. Vasoactive drug infusion (1 drug)	○yes ○no
n. Platelet transfusion	○yes ○no	n. Continuous antiarrythmia infusions	○yes ○no
o. IABP (Intra Aortic Balloon Pressure)	○yes ○no	o. Cardioversion for arrythmia (not defibrillation)	○yes ○no
p. Emergency operative procedures (within past 24 h)	○yes ○no	p. Hypothermia blanket	○yes ○no
q. Lavage of acute GI bleeding	○yes ○no	q. Arterial line	○yes ○no
r. Emergency endoscopy or bronchoscopy	○yes ○no	r. Acute digitalization - within 48 h	○yes ○no
s. Vasoactive drug infusion (> 1 drug)	○yes ○no	s. Measurement of cardiac output by any method	○yes ○no
		t. Active diuresis for fluid overload or cerebral edema	○yes ○no

LOGISTIC ORGAN DYSFUNCTION SCORE:

Logistic organ dysfunction score was developed in 1996, using datas collected from various ICU(77). A score was made with the evaluation of 6 organ systems and 12 variables were analysed. The grading is between 0 and 5 for each organ. The worst value of score obtained in the first 24 hours of icu stay is documented. Though it is not much useful in serial assessment of patients it can assess improvement or worsening of organ dysfunction.

All these existing severity scoring systems utilise a large number of variables and involves a large number of blood investigations which may not be available in all centres except for a sophisticated ICU set up. In an emergency it is difficult to do all such investigations and do a detailed assessment. Also it is so costly to follow up patients with all such investigations. This warranted the need for a simplified scoring system for easy evaluation of patients.

SOFA SCORING SYSTEM:

The SOFA score was developed in 1994, by the European Society of Intensive Care and Emergency Medicine, to provide a means to describe the degree of organ failure in individuals and groups of ICU patients. Vincent et al published the SOFA score and proved that infected patients had more risk of organ dysfunction than the non infected (78)

Table 3 Sequential organ failure assessment score

Organ system	Score				
	0	1	2	3	4
Respiratory PaO_2/FiO_2	> 400	≤ 400	≤ 300	≤ 200	≤ 100
Renal creatinine (μmol/L)	≤ 110	110-170	171-299	300-440 urine output ≤ 500 mL/d	> 440 urine output < 200 mL/d
Hepatic bilirubin (μmol/L)	≤ 20	20-32	33-101	102-204	> 240
Cardiovascular hypotension	No hypotension	MAP < 70 mmHg	Dopamine ≤ 5[1] Dobutamine (any dose)	Dopamine > 5[1] or epinephrine ≤ 0.1[1] or norepinephrine ≤ 0.1[1]	Dopamine > 15[1] or epinephrine > 0.1[1] or norepinephrine > 0.1[1]
Hematologic platelet count (/mL)	> 150	≤ 150	≤ 100	≤ 50	≤ 20
Neurologic Glasgow coma score	15	13-14	10-12	6-9	< 6

SOFA scoring system analyses 6 variables namely

1) Pao2/Fio2 ratio(for respiration)
2) Platelets(for coagulation)
3) Bilirubin (for liver function)
4) Creatinine (for renal function)
5) Glasgow coma scale(to assess level of consciousness)
6) Blood pressure and the need for inotropic support.

A score of 0 to 4 is given for each of these six variables and a score is obtained using sum total value of each of these parameters. The worst values on each day are recorded and organ function total score can thus be monitored over time (79)

The increasing SOFA score and the mean SOFA score are highly useful in assessing prognosis and risk stratification of patients (80 - 82).

Parameters:

Pao2/Fio2 ratio:

It is simply defined as the amount of inspired oxygen that reaches the blood. It is impaired in case of lung injury due to any cause. It is also called carrico index. A Pao2/Fio2 ratio of less than or equal to 200 is required for the diagnosis of acute respiratory distress syndrome according to the AECC criteria(83).

Pao2 is the partial pressure of oxygen in the arterial blood. It is measured in millimetres of mercury(mmHg) or torr units. It is measured by an arterial blood gas analyser(ABG). Normal Pao2 is 75 – 100mmHg.

Fio2 is the percentage of oxygen in the inspired mixture of air. Normal Fio2 in inspired atmospheric air is 0.21(21%). In a mechanical ventilator it is usually set as 30 – 40%. In a mechanically ventilated patient 100% oxygen is not administered due to high risk of oxygen toxicity.

Kerbing and his co workers assessed the clinical relevance of variation in Pao2/Fio2 ratio(84). They demonstrated the clinical utility of this parameter.

The Pao2/Fio2 scores are

Score 0 – more than 400

Score 1 – less than or equal to 400

Score 2 – less than or equal to 300

Score 3 – less than or equal to 200

Score 4 – less than or equal to 100.

Creatinine:

In SOFA scoring serum creatinine values are estimated periodically to assess the renal function over a period of time till the patient is in icu. Creatinine is a breakdown product of creatine phosphate, which is found in muscle. Each day 1-2 % of muscle creatine is converted to creatinine. It is excreted both by glomerular filtration and tubular secretion. Rise in serum creatinine is a marker of damage to nephrons. Normal serum values are 0.7 – 1.2(males) and 0.5 – 1.0(females). Impaired renal function can be due to pre renal, renal or post renal causes. Some of the commonest causes of renal failure are

1) Severe dehydration
2) Acute pyelonephritis
3) Diabetes
4) Hypertension
5) Renal calculi
6) Hemorrhagic fevers
7) Disseminated intravascular coagulation
8) Autoimmune and other connective tissue disorders.

The scores used for creatinine in SOFA score are,

Score 0 – less than 1.2 mg/dl

Score 1 – 1.2 to 1.9 mg/dl

Score 3 – 2.0 to 3.4 mg/dl

Score 4 – 3.5 to 4.9 mg/dl

Score 5 – more than 5 mg/dl

Platelet count:

Platelet count is used as a parameter in SOFA score to assess coagulation function and its impairment during disease states. The coagulation mechanism involves activation, adhesion and aggregation of platelets in response to a stimuli, say an injury or infection. Both platelet

number and function should be adequate for this function to be intact. Coagulation cascade is one of the best understood system in humans (85). Primary hemostasis is mainly due to platelets, which is characterised by formation of platelet plugs (86). Activated platelets release stored granules into the blood. These granules contain

1) Serotonin
2) ADP
3) Platelet activating factor
4) Platelet factor 4
5) Vonwillebrand factor
6) Thromboxane A_2

All these substances when released into the blood stream activate additional platelets. These steps lead on to the activation of various enzymes of coagulation cascade resulting in activation of clotting factors, which is called secondary hemostasis.

Various systemic illness can be associated with a decreased platelet count.

It can be either due to decreased production, increased destruction or impairment of platelet function.

Causes of thrombocytopenia:

1) Vitamin B12 and folate deficiencies
2) Infections like HIV disease
3) Leukemias
4) Disseminated intravascular coagulation
5) Thrombotic thrombocytopenic purpura
6) Viral infections
7) Gram negative septicaemia
8) Heparin induced thrombocytopenia
9) Radiation induced bone marrow suppression
10) Drug toxicity

The scores used for platelet count in SOFA are

Score 0 - >150 × 10^3/mm^3

Score 1 - <150× 10^3/mm^3

Score 2 - <100× 10^3/mm^3

Score 3 - <50× 10^3/mm^3

Score 4 - <20× 10^3/mm^3

Bilirubin:

Bilirubin levels are measured as a marker of liver function. Liver plays a pivotal role in regulating a large number of metabolic pathways in the body. Bile is secreted in the hepatic lobules and it drains ultimately into the bile duct after traversing through canaliculi, small bile ducts and larger bile ducts (87).

It consists of bile acids, phospholipids and unesterified cholesterol. Daily bile output from the liver is 500 – 600ml. It consists of two fractions. Direct or hydrophilic type and indirect or hydrophobic type. Conjugation of indirect to direct fraction takes place in the liver, which is an enzyme mediated process. This whole array of steps in the formation to elimination of bile can be disturbed in disease states. Elevations in bilirubin levels can be used to assess liver function over time, which helps in predicting worsening or improvement of liver function in an ICU patient.

Some of the conditions in which bilirubin levels are raised are,

1) Acute hepatitis
2) Alcoholic liver disease
3) DIC and septicaemia
4) Hepatocellular carcinoma
5) Autoimmune and connective tissue disorders

6) Storage disorders

7) Haemolytic jaundice

8) Obstructive jaundice

9) Congenital liver enzyme abnormalities

10) Massive blood transfusion

Most biologic system in the body gets affected by excess bilirubin in blood. Normal bilirubin levels in blood are 1.0 to 1.5mg/dl (88). Upto 30% of that is direct or conjugated bilirubin, which equals 0.3 mg/dl. It is water soluble. The rest of the fraction is insoluble in water and it is called unconjugated bilirubin.

This is the toxic form of bilirubin, which when accumulates in excess gets deposited in the brain especially in the basal ganglia which may lead to seizures or neurological deficits.

The scores used for bilirubin are

Score 0 - < 1.2 mg/dl

Score 1 – 1.2 to 1.9 mg/dl

Score 2 – 2.0 to 5.9 mg/dl

Score 3 – 6.0 to 11.9 mg/dl

Score 4 - >12 mg/dl

Glasgow coma scale:

It gives a reliable and objective way of recording the conscious state of a person. It is easy to use both for the medical and paramedical personnel for initial as well as continuing medical assessment in an ICU. It has value in predicting ultimate outcome. Three types of responses are assessed.

GCS scale was used initially only for head injury patients. Now it is being used both for acute medical and trauma patients. It is also being used to monitor patients in ICU in a seriously ill state (89). The scale was published in 1974 by Graham Teasdale and Bryan J. Jennett, at the University of Glasgow Institute of Neurological Sciences. Both of them were neurosurgeons.

Glasgow Coma Scale		
Eye Response	Open Spontaneously	4
	Open to Verbal command	3
	Open in response to pain	2
	No response	1
Verbal Response	Talking / Orientated	5
	Confused speech / Disorientated	4
	Inappropriate Words	3
	Incomprehensible sounds	2
	No response	1
Motor Response	Obeys commands	6
	Localizes pain	5
	Withdraws from pain	4
	Abnormal flexion	3
	Extension	2
	No response	1

The highest possible score is 15, that is in a fully awake person. The lowest possible score is 3, which means deep coma or death.

The scores used for GCS in SOFA are

Score 0 – 15

Score 1 – 13 to 14

Score 2 – 10 to 12

Score 3 – 6 to 9

Score 4 - <6

Blood pressure:

"There is no doubt that proper functioning of our pipes and pumps does have an immediate urgency well beyond that of almost any of our other bits and pieces".

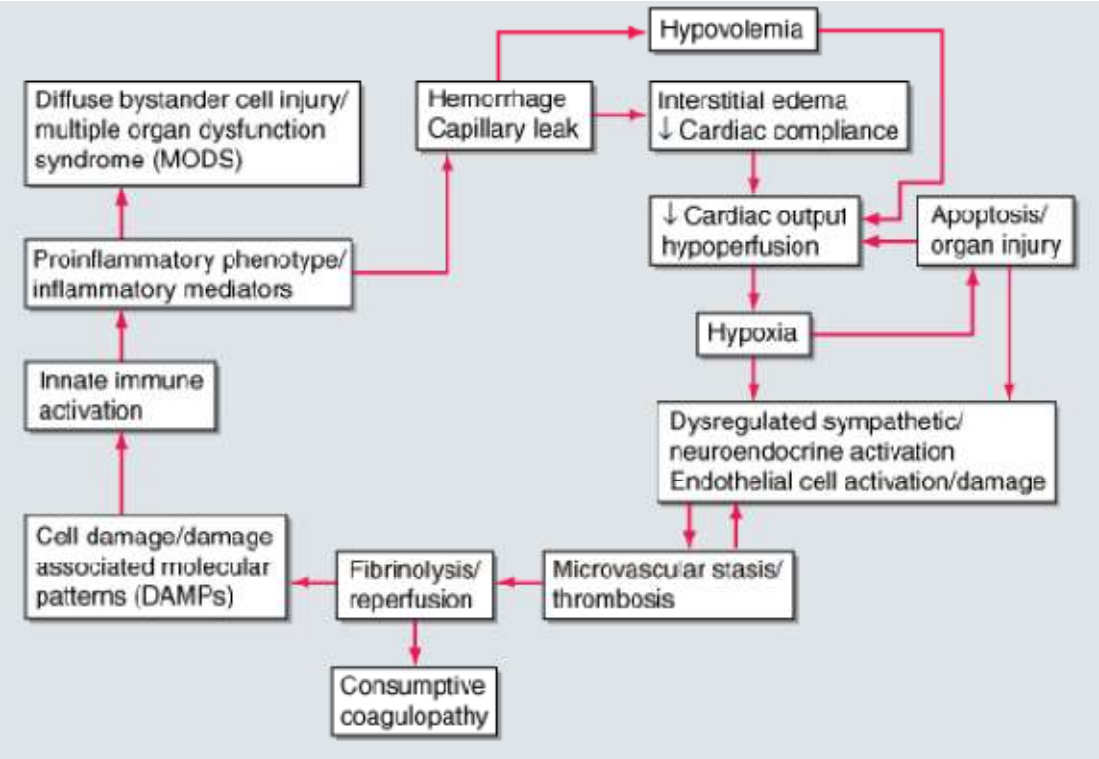
- Steven vogel (vital circuits,1992)

Hypotension and shock may occur as a final consequence of any organ dysfunction. Maintaining an adequate blood pressure is essential for perfusion and oxygenation of vital organs. In short, shock is a clinical syndrome resulting from inadequate tissue perfusion of any cause, resulting in an imbalance between the requirement and supply of oxygen, causing cellular dysfunction. This goes on and on like a vicious cycle resulting in cellular death and multi organ dysfunction.

In an ICU setting cardio respiratory complications are the most common cause of circulatory collapse and shock

Classification of shock:

Hypovolemic	Septic
Traumatic	Hyperdynamic(early)
Cardiogenic	Hypodynamic(late)
Intrinsic	Neurogenic
Compressive	Hypoadrenal



SHOCK INDUCED VICIOUS CYCLE

The scores used for blood pressure in SOFA are

Score 0 – No hypotension

Score 1- Mean arterial pressure <70

Score 2 – dopamine infusion </= 5 or requiring dobutamine

Score 3 – dopamine infusion >/= 5 or requiring nor epinephrine </=0.1

Score 4 – dopamine infusion > 15 or or requiring nor epinephrine 0.1

MATERIALS AND METHODS

Study design:

Prospective observational cohort study

Study group:

Patients admitted to the intensive medical care unit of Coimbatore medical college hospital

Study duration:

One year (May 2013 – May 2014)

Inclusion criteria:

- Age > 15 years
- Patients admitted to the intensive care unit with suspected sepsis/multiorgan dysfunction

Exclusion criteria:

- Age < 15 years
- Patients with less than 48 hours of ICU stay
- Patients who are extremely moribund, who may not survive for more than 48 hours
- Pregnant patients

Sample size:

A total of 100 patients admitted to Coimbatore medical college ICU were studied

Consent:

Informed consent will be taken as per the standard procedures in the institution

Ethical clearance:

Obtained from the ethical committee of the institution

Procedure:

Critically ill suspected multi organ dysfunction patients admitted will be chosen for study if they fulfil the inclusion criteria and parameters needed to calculate SOFA score will be obtained from a patient on admission, 48 and 96 hours after admission at the same time of the day. The scores will be calculated till day 6 or till mortality occurs whichever is earliest.

Blood Investigations will be taken under aseptic conditions with adequate care and sent to the hospital 24 hours laboratory immediately. All the investigations are done in our hospital and no investigations or

procedure will be done outside the hospital. Any experimental or so far unused materials or methods will not be used on the patients.

Serum bilirubin will be calculated using an auto analyser using the method of malloy and evelyn.

ABG was done using ion selective electrode in an ABG analyser

Platelet count was done using sysmex KX21.3 which is an automated cell count analyser, in clinical pathology lab.

Statistical analysis:

Data will be analysed using SPSS software version 17.

RESULTS

Survivors and non survivors:

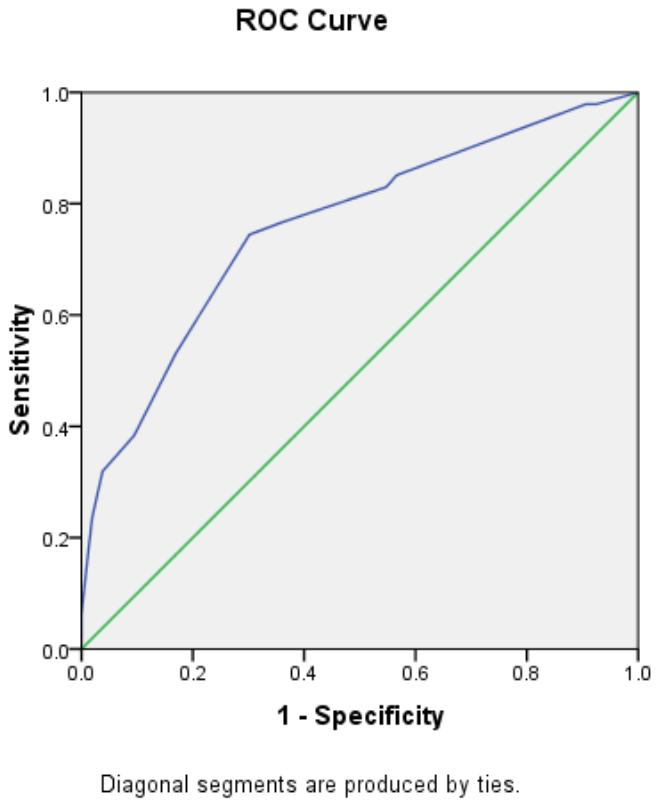
Among the 100 patients involved in the study 53% survived and 47% succumbed to their illness. The minimum age of the person enrolled in the study was 17 and the maximum age was 85.

SOFA score on admission:

SOFA score	Survivors	Non survivors	Total
6 – 7	5	1	6
8 – 9	19	7	26
10 – 11	13	4	17
12 and above	16	35	51
Total	53	47	100

The minimum SOFA score of the patients admitted was 6. Hence the data column starts with values of 6 and above. This table shows that there is a sharp rise in non survivors at a SOFA score above 12.

ROC curve for admission SOFA



Area Under the Curve

Test Result Variable(s): SOFAADMISSION

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.760	.048	.000	.665	.855

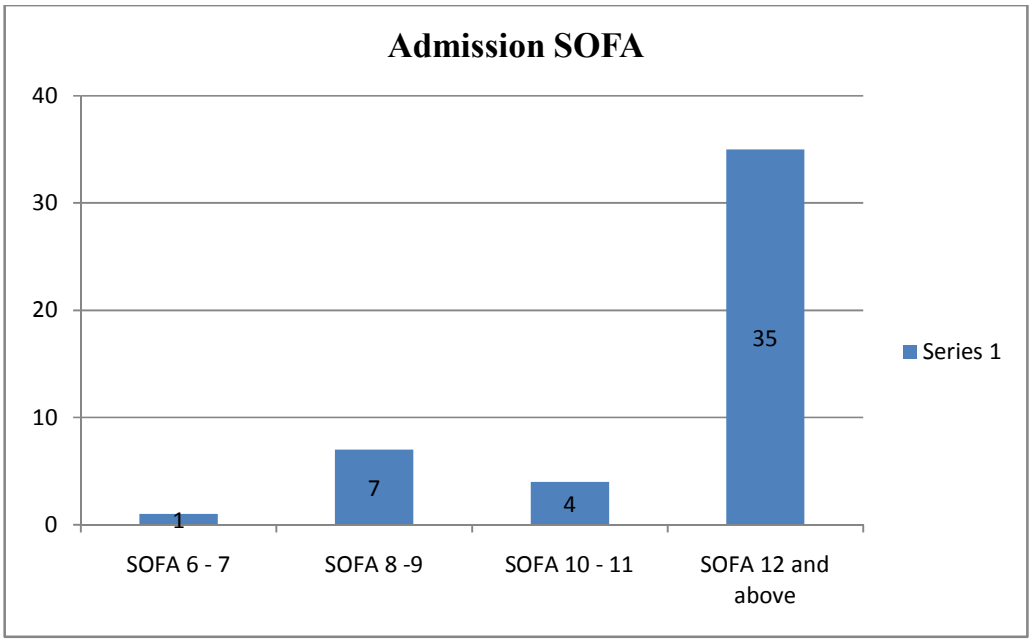
The test result variable(s): SOFAADMISSION has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Bar chart:

No. of deaths



SOFA score

The minimum admission SOFA score of patients in this study is 6. Among the 6 patients who had this score 1 patient expired. That is, the mortality rate is 16.7 %. Among the 61 patients who had an admission SOFA score of 12 and above 35 patients expired escalating the mortality rate to 68.6 %.

SOFA at 48 hours for non survivors:

SOFA score	No. of Non survivors
8 – 9	3
10 - 11	4
12 and above	40

At 48 hours the minimum SOFA score observed among the study population is 8. Hence the data column starts with 8 and above.

Area Under the Curve

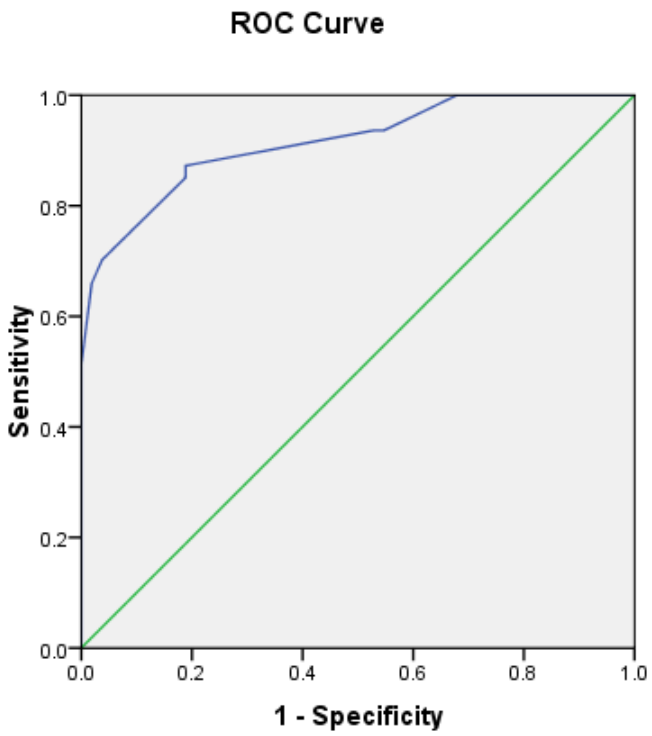
Test Result Variable(s):SOFA48Hr			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
.914	.028	.000	.859	.970

The test result variable(s): SOFA48Hr has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

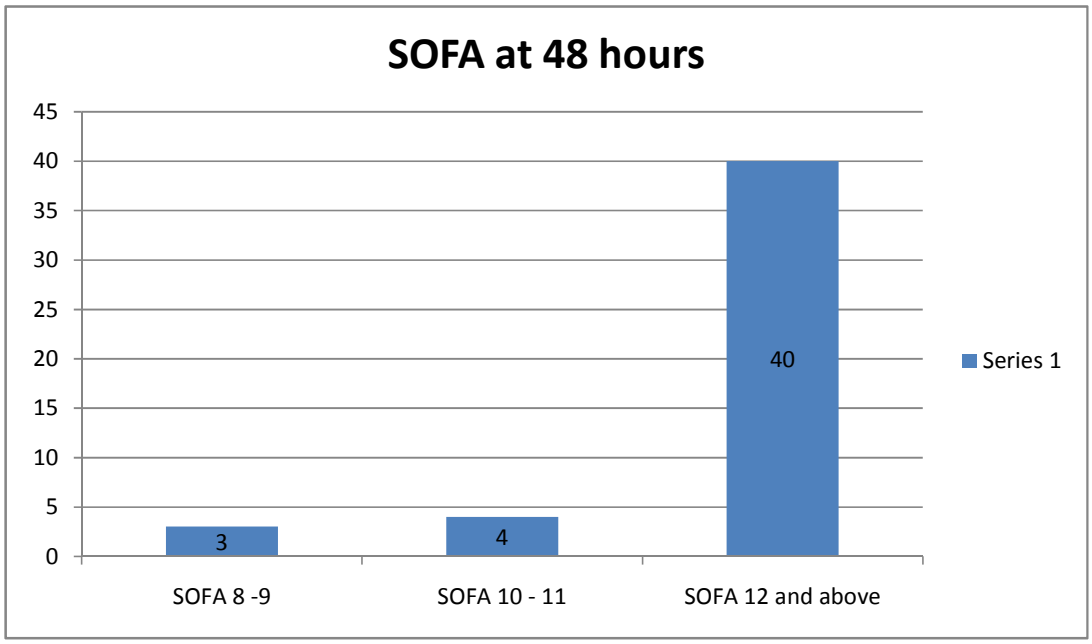
a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

ROC curve for SOFA at 48 hours:



No. Of deaths



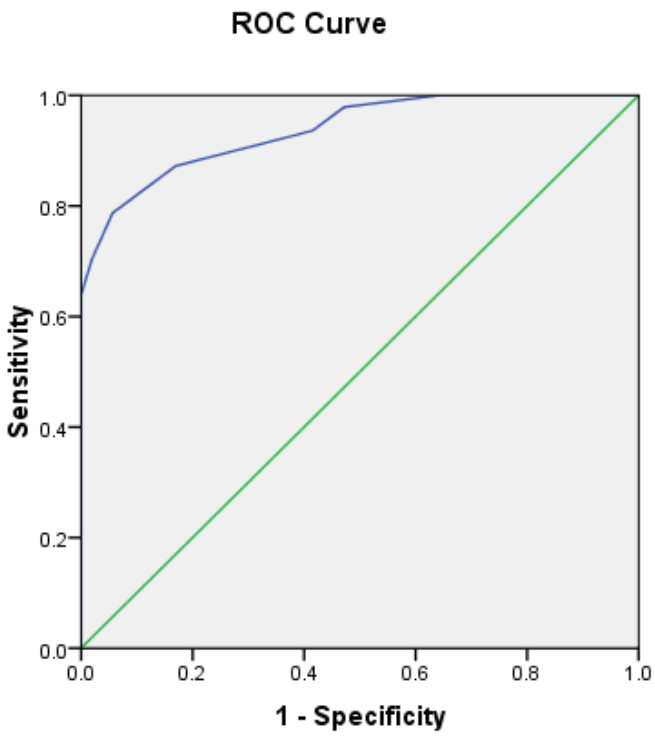
SOFA score

This picture shows that a SOFA score of 12 and above at 48 hours of admission shows an increase in the number of non survivors. The minimum SOFA score of the study population at 48 hours is 8. Among the 47 non survivors, 3 patients had these minimum score. Patients who had a score of 12 and above were 40.

SOFA score at 96 hours for non survivors :

SOFA score	No. Of non survivors
8 – 9	3
10 – 11	3
12 and above	41

ROC curve at 96 hours:



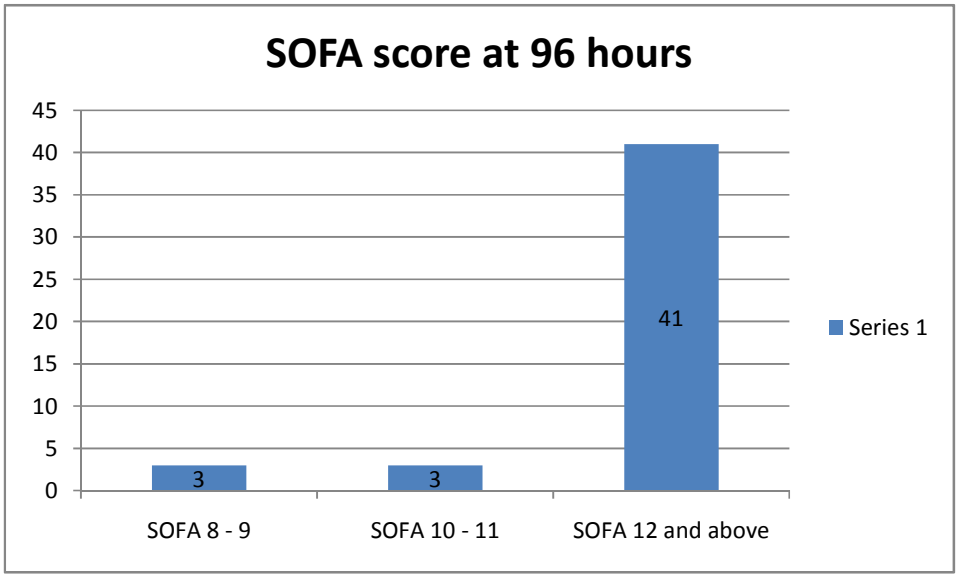
Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s):SOFA96HR			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
.937	.023	.000	.892	.982

Bar diagram:

No. Of deaths



SOFA score

This chart depicts that survival rate is reduced when the SOFA score increases above 12, at 96 hours of admission. At 96 hours 41 out of the 47 patients expired, had a score of 12 and above.

Delta SOFA:

It is the difference between the subsequent SOFA scores. Δ SOFA 48 is the difference between admission score and the score at 48 hours. ΔSOFA 96 is the difference between the score at 48 hours and 96 hours.

SOFA score 48 hour changes:

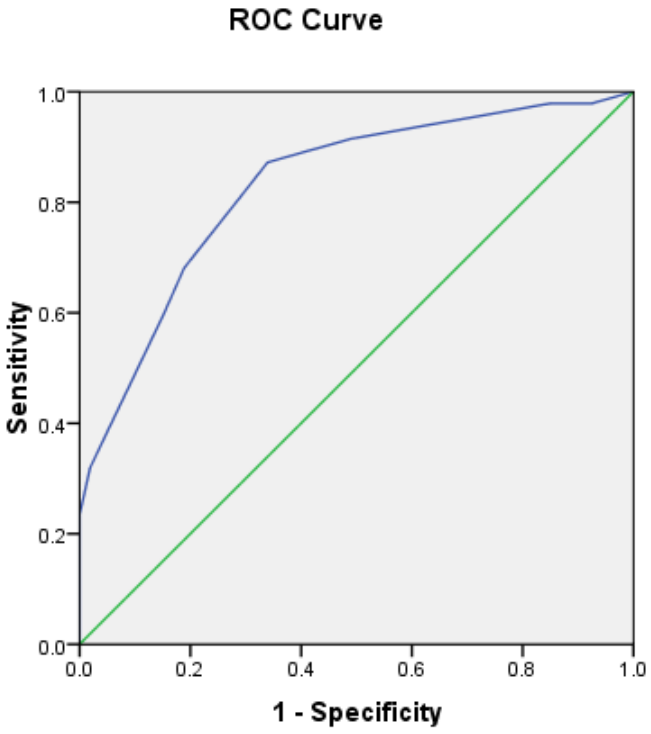
The patient data is analysed as those who decreased, unchanged and increased from the initial score respectively, and the outcome is analysed.

Δ SOFA 48	**Survivors**	**Non survivors**
Decreased	35	6
Unchanged	8	9
Increased	10	32

Area Under the Curve

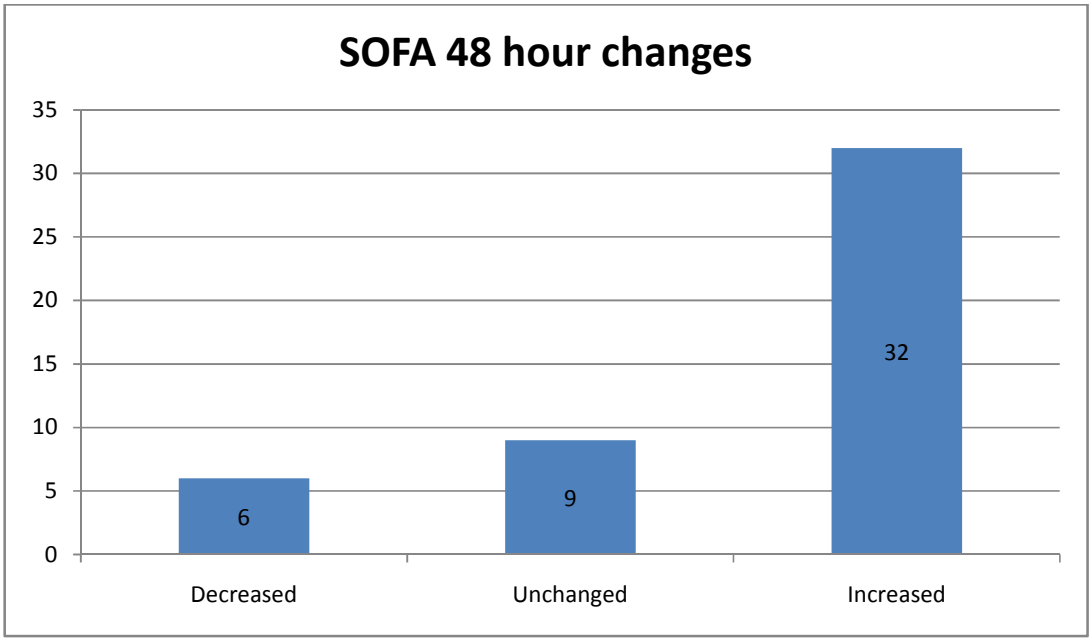
Test Result Variable(s):SOFA48 difference			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
.830	.041	.000	.749	.910

SOFA 48 hour changes:



Diagonal segments are produced by ties.

No. Of deaths



SOFA score

These data depicts that when the SOFA score is increased from admission to 48 hours, there is an increase in mortality. On contrary the mortality rate has decreased when the score falls. Among the 47 non survivors 32 (68.08%) had an increase in their Δ48 scores.

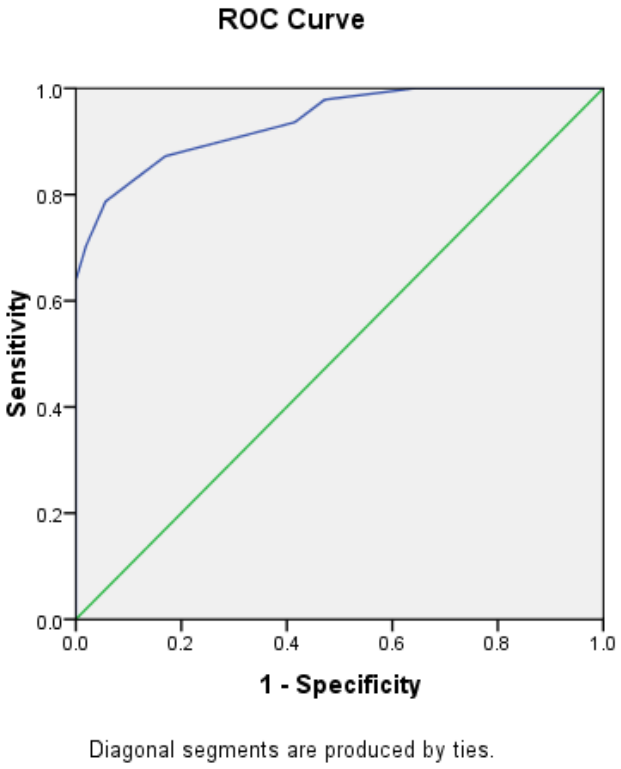
SOFA score 96 hour changes:

The patient data is analysed as those who decreased, unchanged and increased from the initial score respectively, and the outcome is analysed.

Δ SOFA 96	Survivors	Non survivors
Decreased	39	7
Unchanged	7	2
Increased	7	38

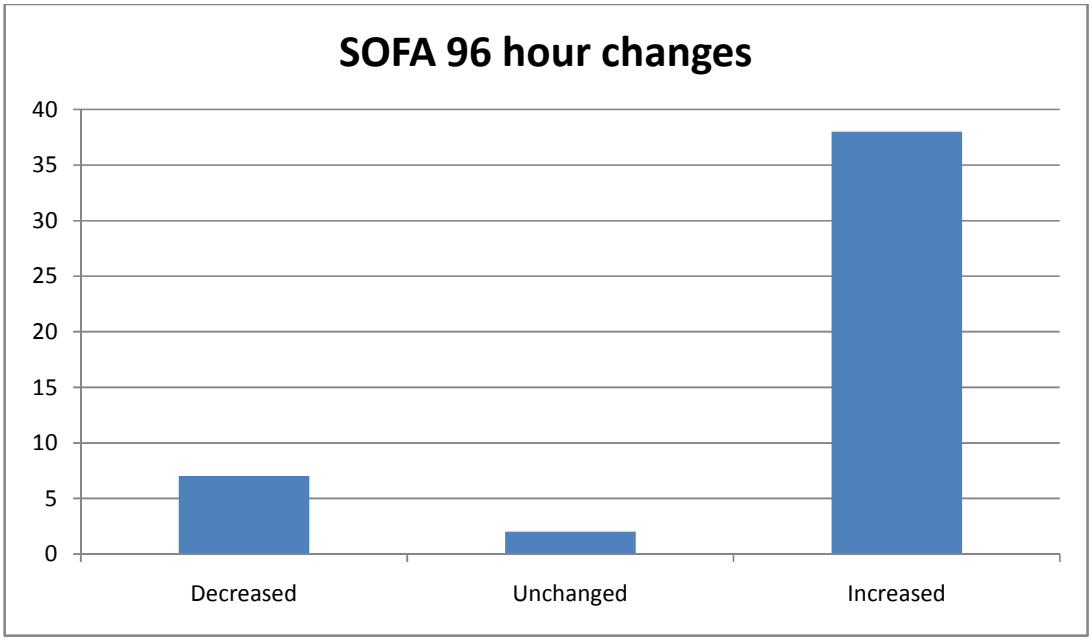
SOFA 96 hour changes:

Figure 6:



Bar chart:

No. Of deaths

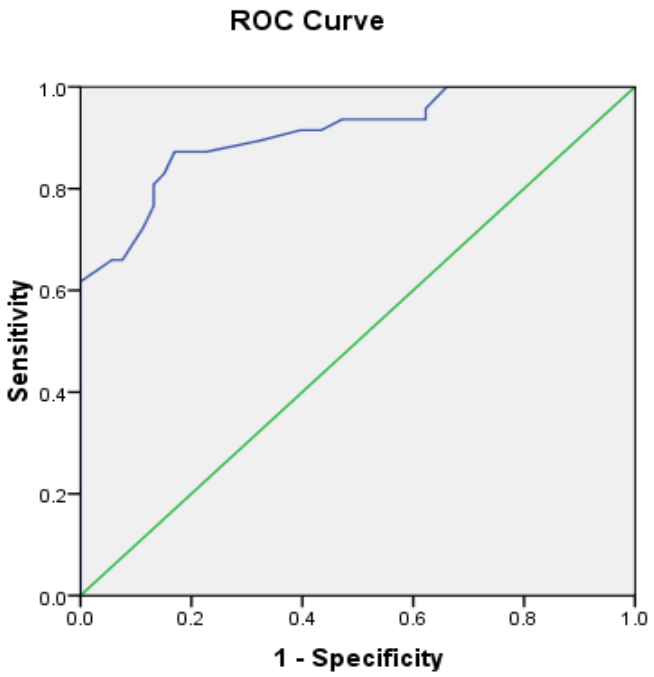


SOFA score

This chart depicts mortality rate is increased when the SOFA score is increased from admission to 96 hours. On contrary, the mortality rate has decreased when the score falls. Among the 47 non survivors 38 (80.85%) had an increase in their Δ 96 scores.

Mean SOFA:

Mean SOFA calculates the average value of the prognostic score during the entire ICU stay of the patient.



Diagonal segments are produced by ties.

Test Result

Variable(s):MEANSOFA

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
.908	.029	.000	.851	.966

Coordinates of the Curve

Test Result Variable(s): MEANSOFA

Positive if Greater Than or Equal To[a]	Sensitivity	1 – Specificity
4.3333	1.000	1.000
5.6667	1.000	.962
6.3333	1.000	.925
7.0000	1.000	.755
7.5000	1.000	.660
7.8333	.979	.642
8.1667	.957	.623
8.5000	.936	.623
8.8333	.936	.491
9.1667	.936	.472
9.5000	.915	.434
10.0000	.915	.396
10.5000	.894	.321
10.8333	.872	.226
11.167	**0.87**	**0.17**
11.5000	.830	.151
11.8333	.809	.132
12.1667	.766	.132
12.5000	.723	.113
12.8333	.660	.075
13.1667	.660	.057
13.5000	.617	.000
13.8333	.574	.000
14.1667	.532	.000
14.5000	.489	.000
14.8333	.404	.000
15.1667	.383	.000
15.5000	.319	.000
16.1667	.277	.000
16.8333	.213	.000

17.1667	.191	.000
17.5000	.149	.000
18.0000	.106	.000
18.5000	.064	.000
20.0000	.021	.000
22.3333	.000	.000

The test result variable(s): MEANSOFA has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

These data shows that, a mean SOFA score of 11 and above is an excellent predictor of mortality, above which the number of non survivors increase.

Total SOFA:

It is the sum total of all the scores obtained from an individual patient during his hospital stay. It gives information about the severity of the illness since gives the total worst score of all organs.

Area under the curve: Test Result Variable(s):TOTALSOFA Table 11:			Asymptotic 95% Confidence Interval	
Area	Std. Error[a]	Asymptotic Sig.[b]	Lower Bound	Upper Bound
.908	.029	.000	.851	.966

Coordinates of the Curve

Test Result Variable(s): TOTALSOFA

Positive if Greater Than or Equal To[a]	Sensitivity	1 – Specificity
15.0000	1.000	1.000
17.0000	1.000	.962
19.0000	1.000	.925
21.0000	1.000	.755
22.5000	1.000	.660
23.5000	.979	.642
24.5000	.957	.623
25.5000	.936	.623
26.5000	.936	.491
27.5000	.936	.472
28.5000	.915	.434
30.0000	.915	.396
31.5000	.894	.321
32.5000	.872	.226
33.500	**0.87**	**0.17**
34.5000	.830	.151

35.5000	.809	.132
36.5000	.766	.132
37.5000	.723	.113
38.5000	.660	.075
39.5000	.660	.057
40.5000	.617	.000
41.5000	.574	.000
42.5000	.532	.000
43.5000	.489	.000
44.5000	.404	.000
45.5000	.383	.000
46.5000	.319	.000
48.5000	.277	.000
50.5000	.213	.000
51.5000	.191	.000
52.5000	.149	.000
54.0000	.106	.000
55.5000	.064	.000
60.0000	.021	.000
65.0000	.000	.000

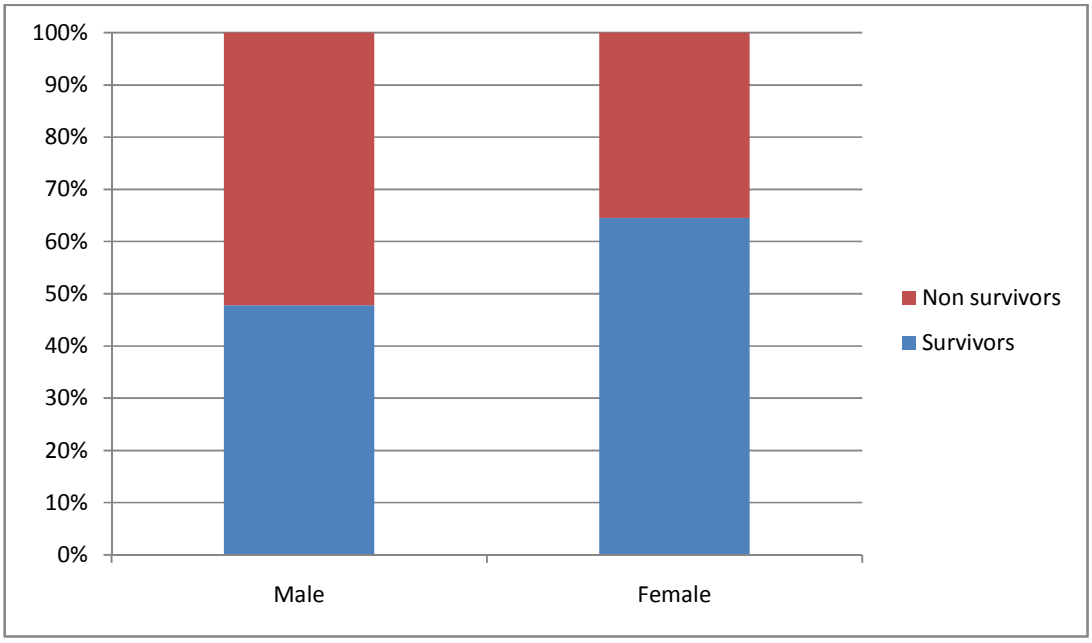
The test result variable(s): TOTALSOFA has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

These data depict that a total SOFA score of 33 and above is an excellent predictor of mortality, above which the number of non survivors increase.

Outcome based on sex:

Sex	Survivors	Non survivors	Total
Male	33	36	69
Female	20	11	31
Total	53	47	100

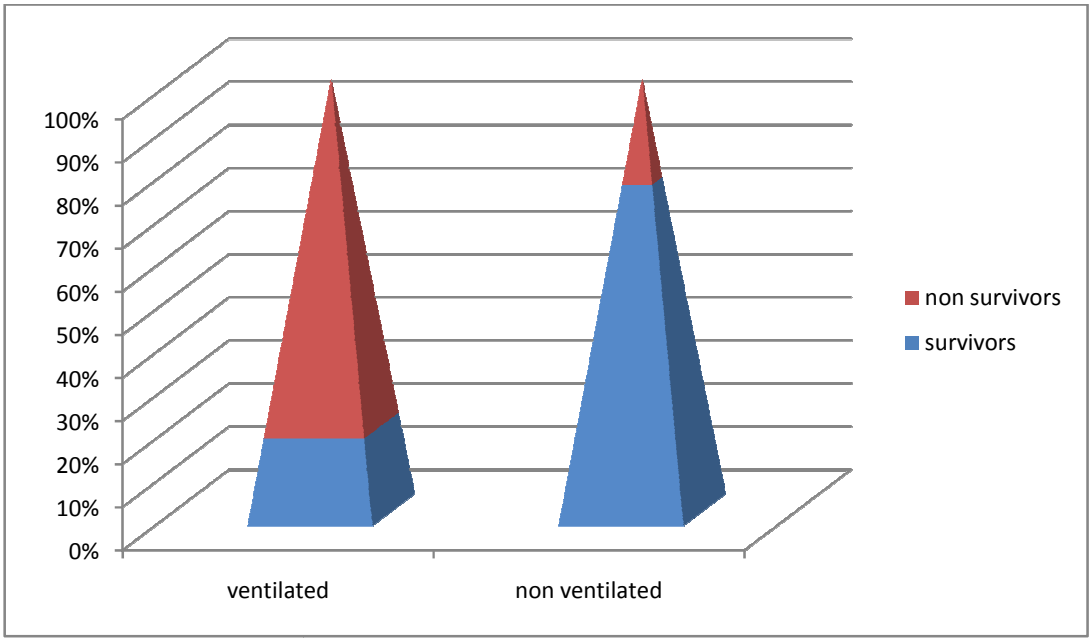


Out of 69 male patients, 36 (52.2%) patients expired and out of 31 female patients, 11(35.5%) patients expired.

Outcome for ventilator support:

Mechanical Ventilation status	Survivors	Non survivors
Ventilated	8	33
Non ventilated	45	14

Graphic representation:



Statistical significance of outcomes related to need for mechanical ventilation:

Chi-Square Tests					
	Value	**df**	**Asymp. Sig. (2-sided)**	**Exact Sig. (2-sided)**	**Exact Sig. (1-sided)**
Pearson Chi-Square	31.285[a]	1	.000		
Continuity Correction[b]	29.048	1	.000		
Likelihood Ratio	33.141	1	.000		
Fisher's Exact Test				.000	.000
Linear-by-Linear Association	30.972	1	.000		
N of Valid Cases[b]	100				
a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 19.27.					

Among the 41 patients ventilated 33 (80.5%) expired and among the 59 patients who did not require ventilator support 14 (23.7%) expired.

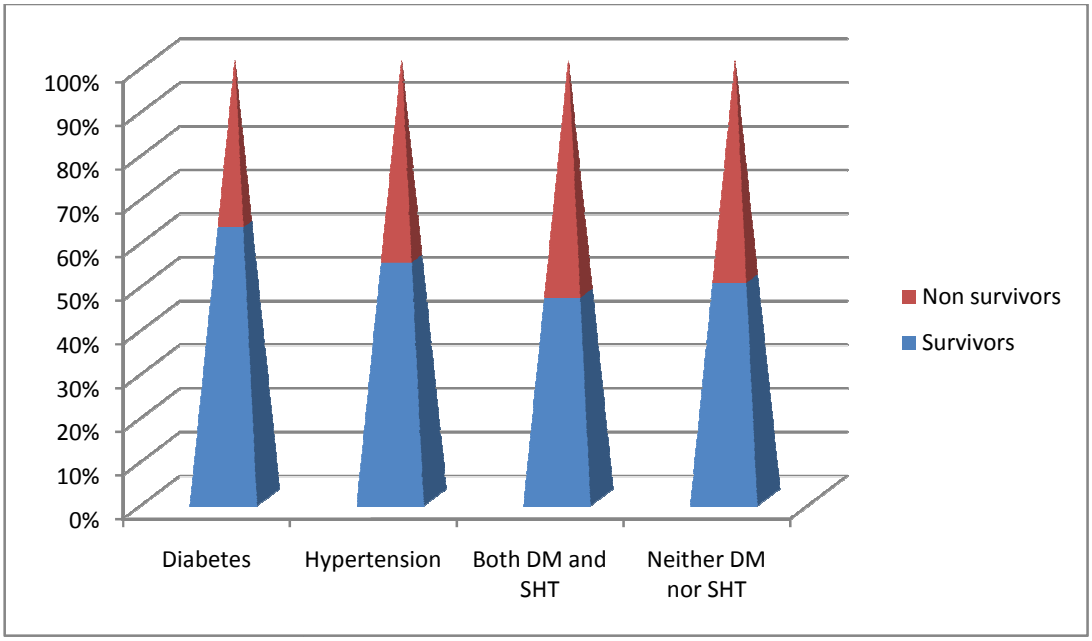
Outcome related to comorbidities:

Two comorbid illness are taken in this study. The patients were categorised as having

1) Diabetes
2) Hypertension
3) Both diabetes and hypertension
4) Neither diabetes nor hypertension

Comorbid illness	survivors	Non survivors
Diabetes	15	9
Hypertension	6	5
Both DM and SHT	7	8
Neither DM nor SHT	25	25

Graphic representation:

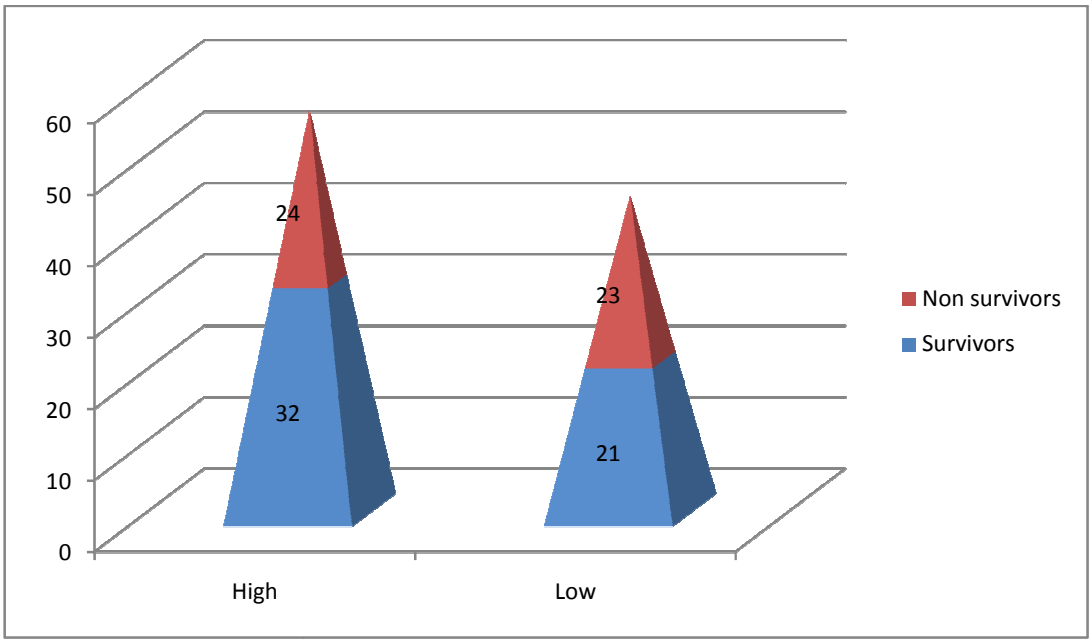


Among the 47 non survivors, 9 are diabetics, 5 are hypertensives, 8 are both diabetic and hypertensives, 25 are neither diabetic nor hypertensive. These comorbidities are not found to have any relationship with outcome in our study.

Outcomes in relation to socioeconomic status:

Socioeconomic status	Survivors	Non survivors
High	32	24
Low	21	23
Total	53	47

Socioeconomic status



Chi-Square Tests:

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.877[a]	1	.349		
Continuity Correction[b]	.540	1	.463		
Likelihood Ratio	.878	1	.349		
Fisher's Exact Test				.421	.231
Linear-by-Linear Association	.868	1	.351		
N of Valid Cases[b]	100				

Patients were segregated into low and high socioeconomic groups based on modified kuppusamy scale. Out of the 56 patients belonging to high socioeconomic group 24 (42.9%) expired. Out of the 44 patients in the low socioeconomic group 23(52.3%) expired.

DISCUSSION

Since the cost of health care is increasing day to day, assessment of a patient's prognosis is vital during the course of treatment. Outcome prediction gains importance in this regard. So scoring systems have been used to predict this. SOFA scoring system, because of its simplicity and easy applicability, has been widely used in ICU setting. This system has also been evaluated in many ICUs and found to be useful as a simple bedside tool.

In our study sex of the patient did not play a significant role in influencing mortality. The morbidity and mortality is purely related to the underlying disease state.

Also comorbidities like diabetes and hypertension did not influence the outcome much, since there is no much statistical significance.

But, the need for mechanical ventilation clearly predicted mortality outcome since, the patients who were ventilated showed a higher mortality rate compared to those who did not require ventilator support, as evidenced by the statistically significant p value < 0.001.

Patients belonging to low socioeconomic status showed higher mortality rate(52.3%) compared to their counterparts belonging to high

socioeconomic state(42.9%). Though the values are not statistically significant in our study, to prove the association.

There is a significant increase in mortality rate when the SOFA score is above 12. There is a steep rise in the mortality curve at this value. Admission SOFA, 48 hours SOFA and 96 hours SOFA are all statistically significant with a p value < 0.001.

Delta SOFA which is the difference in values over a period of time is also statistically significant in our study. There is a strong evidence that, patients whose delta SOFA values when increased from the previous value, there is a greater chance that the patient may succumb to his illness.

Mean SOFA value also proved to be an independent predictor of mortality. A value of more than 11 showed a sharp rise in mortality.

Total SOFA score is also statistically significant in predicting mortality, irrespective of the disease state. A total SOFA score of more than 33.5 is associated with increased mortality.

In summary SOFA score is very useful in predicting mortality in critically ill patients, since there is a strong correlation between a rise in the score and mortality in all stages of admission.

CONCLUSION

- There is a strong association between rise in SOFA score and mortality.
- Mechanically ventilated patients have a high risk of mortality compared to non ventilated patients.
- There is no significant association between comorbidities like diabetes and hypertension with mortality outcome.

LIMITATIONS OF THE STUDY

- Limited number of subjects involved in the study. A larger study population will give more precise results
- Since the age group varies from 17 to 85, age may influence the outcome which is not considered in our study.
- Only the scores were considered irrespective of the underlying disease. So the mortality outcome of individual disease has not been studied, which might also have influenced the outcome.
- Furthermore, it is only a single centre study. The disease pattern and patient profile may vary across various geographic locations, which in turn may influence the outcome.

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PROFORMA

SERIAL NO: DATE:

NAME: AGE:

SEX: ADDRESS:

OCCUPATION:

DATE OF ADMISSION:

COMPLAINTS:

HISTORY:

DIABETES –

HYPERTENSION -

SMOKING –

ALCOHOL –

CLINICAL EXAMINATION:

PULSE: BLOOD PRESSURE: GCS SCORE:

INVESTIGATIONS:

COMPLETE BLOOD COUNT:

SERUM CREATININE:

SERUM BILIRUBIN:

ABG ANALYSIS:

MECHANICAL VENTILATORY SUPPORT: YES/NO

ADMISSION SOFA: SOFA 48: SOFA 96:

CONDITION AT DISCHARGE: SURVIVOR/ NON SURVIVOR

CONSENT FORM

I hereby agree that, I include myself in the study- "**A Study on Mortality Outcomes in ICU Patients with Sequential Organ Failure Assessment (SOFA) Score"** conducted by **Dr.santhakumar.R.P.S.P**. I have been informed that i would not be facing any adverse health problems due to the study and this will not affect the nature of the treatment at any cost.

I have also been informed about the investigations i will be undergoing during the study period and I have the right to withdraw from the research project, if i feel that i am not ok with the project, at any point of time and it will not affect my further treatment. I also agree to disclose the finding of the examinations protecting the privacy as it is essential to predict the outcome of the study.

Name & Signature of the patient **Name & Signature of the relative**

Place:

Date:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் **சாந்தகுமார்.R.P.S.P.** அவர்கள் மேற்கொள்ளும் **"SOFA SCORE அளவுகோல் கொண்டு தீவிர சிகிச்சை பிரிவு நோயாளிகளின் இறப்பினை கணித்தல்"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடன், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

SOFA score:

	0	1	2	3	4
Respiration PaO_2/FIO_2 (mm Hg) SaO_2/FIO_2	>400	<400 221–301	<300 142–220	<200 67–141	<100 <67
Coagulation Platelets $10^3/mm^3$	>150	<150	<100	<50	<20
Liver Bilirubin (mg/dL)	<1.2	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Cardiovascular[b] Hypotension	No hypotension	MAP <70	Dopamine </=5 or dobutamine (any)	Dopamine >5 or norepinephrine </=0.1	Dopamine >15 or norepinephrine >0.1
CNS Glasgow Coma Score	15	13–14	10–12	6–9	<6
Renal Creatinine (mg/dL) or urine output (mL/d)	<1.2	1.2–1.9	2.0–3.4	3.5–4.9 or <500	>5.0 or <200

MAP - mean arterial pressure, CNS - central nervous system, SaO_2- peripheral arterial oxygen saturation, [a]PaO_2/FIO_2 ratio was used preferentially, If not available, the SaO_2/FIO_2 ratio was used; [b]Vasoactive mediations administered for at least 1 hr (dopamine or norepinephrine ug/kg/min).

LIST OF ABBREVIATIONS

ABG	–	Arterial blood gas analysis
ACCF	–	American college of cardiology foundation
AHA	-	American heart association
APACHE	–	Acute physiology and chronic health evaluation
ARDS	–	Adult respiratory distress syndrome
BEE	–	Basal energy expenditure
CIM	-	Critical illness myopathy
CIN	–	Critial illness neuropathy
CIP	–	Critical illness polyneuropathy
CMAP	–	Compound muscle action potential
EMG	-	Electromyogram
ESC	–	European society of cardiology
ESPEN	–	European society for parenteral and enteral nutrition
FiO2	–	Fraction of inspired oxygen
GBS	–	Guillain Barre Syndrome
GCS	–	Glasgow coma scale
ICU	–	Intensive care unit
ICS	–	Intensive care society
LBBB	–	Left bundle branch block
LLN	–	Lower limit of normal
LODS	–	Logistic organ dysfunction score
MODS	–	Multi organ dysfunction score

MODS	–	Multi organ dysfunction syndrome
MUPs	–	Motor unit potentials
MPM	–	Mortality probability model
NICE	–	National institute for health and clinical excellence
PCI	–	Percutaneous coronary intervention
PEEP	–	Positive end expiratory pressure
r- tPA	–	Recombinant tissue plasminogen activator
SIRS	–	Systemic inflammatory response syndrome
SNAP	–	Sensory neuron action potential
SOFA	–	Sequential organ failure assessment
UTI	–	Urinary tract infection
VILI	–	Ventilator induced lung injury
WHF	–	World health federation

KEY TO MASTER CHART

ARDS – Adult respiratory distress syndrome

CVA – cerebrovascular accident

CVT – cortical venous thrombosis

COPD – chronic obstructive pulmonary disease

OPC – organophosphorus poisoning

Coding:

A – Survivor

B – Non survivor

R1 – Smoker

R2 - Alcoholic

R3 – Both smoker and alcoholic

R4 – Non smoker and non alcoholic

1 - Diabetic

2 – Hypertensive

3 – Both diabetic and hypertensive

4 – Non diabetic and non hypertensive

X – High socioeconomic group

Y – Low socioeconomic group

MASTER CHART

S.NO	Name	AGE	SEX	DIAGNOSIS	COMORBID ILLNESS	VENTILATOR SUPPORT	SOFA SCORE ON ADMISSION	SOFA SCORE AT 48 HOURS	SOFA SCORE AT 96 HOURS	RISK FACTORS	SOCIOECONOMIC STATUS	OUTCOME
1	GANESH	60	M	OPC POISONING	4	YES	9	11	15	R1	Y	B
2	ARUKKANI	65	F	SNAKE BITE	1	NO	6	6	4	R4	X	A
3	MOOKAYEE	52	F	CVA HEMORRHAGIC STROKE	2	YES	8	10	13	R4	X	B
4	SELVAM	42	M	CHRONIC KIDNEY DISEASE	3	NO	10	12	10	R3	X	A
5	KARUPPAN	42	M	CIRRHOSIS LIVER	4	NO	12	16	18	R2	Y	B
6	LAKSHMI	68	F	YELLOW DYE POISONING	1	YES	10	12	12	R4	X	B
7	SUBBAN	23	M	SNAKE BITE	4	YES	8	14	14	R4	X	B
8	VADIVU	70	F	MYOCARDIAL INFARCTION	3	YES	12	10	10	R4	Y	A
9	KARTHIKEYAN	45	M	CVA ISCHAEMIC STROKE	3	YES	13	15	15	R1	Y	B
10	POONGODI	19	F	ALUMINIUM PHOSPHIDE POISONING	1	NO	16	20	20	R4	X	B
11	MUNIYAN	60	M	CIRRHOSIS LIVER	1	NO	8	6	6	R3	Y	A
12	MOOKANDI	59	M	MYOCARDIAL INFARCTION	3	NO	8	4	4	R1	X	A
13	LEELAVATHY	60	F	MYOCARDIAL INFARCTION	2	YES	6	6	6	R4	X	A
14	JENCY	52	F	CHRONIC KIDNEY DISEASE	1	NO	8	10	10	R4	Y	A
15	ALAGUMUTHU	75	M	ULCER FOOT/SEPTICEMIA	1	NO	15	18	20	R1	X	B
16	SURYAPRAKASH	36	M	MYOCARDIAL INFARCTION	1	NO	8	6	6	R3	Y	A
17	BASKAR	52	M	CHRONIC KIDNEY DISEASE	1	NO	15	18	18	R2	Y	B
18	BALAJI	61	M	MYOCARDIAL INFARCTION	3	NO	12	10	10	R3	X	A
19	SENTHIL	18	M	SNAKE BITE	4	NO	10	6	6	R3	Y	A
20	LATHA	70	F	CVA HEMORRHAGIC STROKE	3	YES	8	10	10	R4	X	B
21	BALU	48	M	PARAQUAT POISONING	4	YES	8	14	20	R3	Y	B
22	KALAI	19	F	OPC POISONING	4	YES	12	16	16	R4	Y	B
23	KARUNAISELVAN	32	M	CIRRHOSIS LIVER	4	NO	10	8	8	R2	X	A
24	MUTHU	23	M	CIRRHOSIS LIVER	4	NO	12	10	9	R3	X	A
25	KATHIRAVAN	50	M	YELLOW DYE POISONING	4	NO	8	6	6	R4	Y	A

26	KUMARESAN	65	M	MYOCARDIAL INFARCTION	1	NO	12	10	10	R4	X	B
27	DURAI	38	M	OPC POISONING	4	NO	10	14	16	R3	Y	B
28	RAJESHWARI	37	F	CVA ISCHAEMIC STROKE	1	NO	8	10	8	R4	Y	A
29	KAVITHA	35	F	CHRONIC KIDNEY DISEASE	4	NO	11	10	10	R4	X	A
30	JEEVA	23	F	PYELONEPHRITIS	4	NO	14	12	12	R4	X	A
31	RAJA	41	M	CIRRHOSIS LIVER	4	NO	16	18	19	R3	Y	B
32	SARAVANAN	50	M	OPC POISONING	1	YES	15	20	20	R1	X	B
33	GOWRISANKAR	68	M	GANGRENE FOOT	1	YES	12	16	18	R3	X	B
34	RAMU	17	M	SNAKE BITE	4	NO	13	10	10	R4	Y	A
35	BANGARU	46	F	MYOCARDIAL INFARCTION	1	NO	15	12	12	R4	X	A
36	KANAGARAJ	52	M	CIRRHOSIS LIVER	4	YES	11	10	10	R2	X	A
37	VENKATESH	35	M	CVA HEMORRHAGIC STROKE	2	YES	11	15	15	R1	Y	B
38	SUBBU	60	M	MYOCARDIAL INFARCTION	3	NO	8	8	8	R3	Y	A
39	KATHAYEE	55	F	MYOCARDIAL INFARCTION	1	NO	8	10	8	R4	Y	A
40	KUMARESAN	59	M	CVA ISCHAEMIC STROKE	2	NO	12	10	10	R1	X	A
41	IDUMBAN	48	M	CIRRHOSIS LIVER	4	NO	8	6	6	R3	X	A
42	SUKUMAR	35	M	OPC POISONING	4	YES	12	10	12	R3	Y	A
43	SAVEETHA	28	F	CVT	4	YES	12	13	13	R4	X	B
44	RAJASEKAR	35	M	PNEUMONIA	1	YES	13	10	10	R1	X	A
45	VINOTH	54	M	CHRONIC KIDNEY DISEASE	2	YES	10	12	14	R3	Y	B
46	SARAVANAN	25	M	BACTERIAL MENINGITIS	4	NO	10	8	8	R2	X	A
47	RAMESH	46	M	CIRRHOSIS LIVER	4	YES	15	15	16	R1	Y	B
48	RAHUMAN	70	M	MYOCARDIAL INFARCTION	3	NO	13	14	14	R4	X	B
49	KATHEEJA	36	F	OPC POISONING	4	YES	13	14	16	R4	X	B
50	PRADEEP	50	M	CVA ISCHAEMIC STROKE	2	NO	8	6	6	R3	Y	A

51	GUNASEKAR	60	M	CVA HEMORRHAGIC STROKE	2	YES	12	16	16	R1	X	B
52	SENBAHAM	38	F	SNAKE BITE	4	NO	8	7	7	R4	X	A
53	NATARAJAN	55	M	PNEUMONIA	3	YES	14	16	17	R3	X	B
54	SANGUMUTHU	40	M	CHRONIC KIDNEY DISEASE	3	NO	10	9	8	R4	Y	A
55	RAVISANKAR	52	M	PARAQUAT POISONING	4	YES	16	16	18	R3	X	B
56	GOVINDAN	66	M	CVA HEMORRHAGIC STROKE	3	YES	13	12	12	R1	Y	B
57	MANISHARMA	56	M	CIRRHOSIS LIVER	4	YES	14	15	16	R3	Y	B
58	GAYATHRI	60	F	MYOCARDIAL INFARCTION	1	NO	11	10	10	R4	X	A
59	SELVARAJ	46	M	ALUMINIUM PHOSPHIDE POISONING	4	YES	16	19	20	R2	X	B
60	SAROJA	63	F	CHRONIC KIDNEY DISEASE	1	NO	13	10	10	R4	Y	A
61	INBARAASU	72	M	AORTIC DISSECTION	2	NO	8	8	9	R1	Y	B
62	RASIMUTHU	46	M	ASPIRATION PNEUMONIA	1	NO	8	6	6	R3	X	A
63	KOTHAI	59	F	TB MENINGITIS	4	YES	10	8	8	R4	Y	A
64	ANBUMANI	61	M	SNAKE BITE	4	NO	8	10	10	R2	X	A
65	BALAMANI	63	F	CIRRHOSIS LIVER	4	NO	12	12	10	R2	Y	B
66	SUNDARAVEL	70	M	OPC POISONING	4	YES	16	12	12	R2	Y	B
67	PRAVEEN	19	M	SICKLE CELL ANAEMIA	4	NO	8	7	7	R4	Y	A
68	RAJAN	25	M	MYOCARDIAL INFARCTION	4	NO	8	6	6	R3	X	A
69	VIJAYSANKAR	30	M	H1N1 PNEUMONIA	1	YES	12	16	16	R1	Y	B
70	PRATHAP	66	M	CHRONIC KIDNEY DISEASE	2	NO	7	10	9	R2	Y	A
71	IRUTHAYAMARY	85	F	COPD COR PULMONALE	4	YES	16	18	18	R4	X	B
72	SATHISH	30	M	PARAQUAT POISONING	4	YES	20	22	22	R3	Y	B
73	KATHAMUTHU	64	M	PULMONARY TB	1	NO	10	6	6	R1	X	A
74	LAXMIPRIYA	67	F	DIABETIC KETOACIDOSIS	1	NO	16	12	12	R4	X	A
75	GEETHAMATHI	47	F	ARDS/SEPSIS	4	YES	20	18	18	R2	X	B

76	SANTHOSH	56	M	CIRRHOSIS LIVER	4	N0	16	18	18	R3	Y	B
77	PRIYA	18	F	OLEANDER SEED POISONING	4	NO	6	6	6	R4	Y	A
78	GOMATHI	60	F	DIABETES MELLITUS/SEPTICEMIA	1	NO	12	14	14	R4	Y	A
79	JAMESON	50	M	AIDP - GUILLAIN BARRE SYNDROME	4	YES	18	16	16	R4	X	B
80	PANDIYAN	40	M	PNEUMOTHORAX	4	NO	10	8	8	R1	X	A
81	RAJARATHINAM	75	M	SUBARACHNOID HEMORRHAGE	3	YES	12	14	16	R3	Y	B
82	KUMARAVEL	64	M	CAHD/CARDIAC FAILURE	2	NO	13	12	12	R3	X	A
83	THANGARAJ	44	M	CIRRHOSIS LIVER/HEPATIC ENCEPHALOPA	4	NO	13	16	18	R2	X	B
84	XAVIER	50	M	SEIZURE DISORDER	4	YES	12	12	8	R4	X	A
85	ELANGO	28	M	ACID POISONING	4	NO	8	8	6	R3	X	A
86	PALANI	17	M	BACTERIAL MENINGITIS	4	YES	14	14	16	R1	Y	B
87	MARUTHACHALAM	35	M	PULMONARY EMBOLISM	4	YES	8	8	8	R2	X	B
88	MARIMUTHU	35	M	ARDS/SEPSIS	4	NO	13	13	12	R3	Y	B
89	KRISHNAKUMAR	70	M	SNAKE BITE	3	YES	6	8	9	R4	X	B
90	HASEENA	40	F	RHEUMATIC HEART DISEASE	4	YES	12	12	13	R4	X	B
91	BOMMAN	60	M	ACUTE MYELOID LEUKEMIA	1	NO	14	12	12	R4	X	A
92	ANBUSELVI	28	F	ACUTE LYMPHOBLASTIC LEUKEMIA	4	NO	8	6	6	R4	Y	A
93	SAKTHIVEL	35	M	ACUTE ALCOHOL INTOXICATION	4	NO	10	10	9	R3	Y	A
94	SELVAMUTHU	45	M	DIABETIC KETOACIDOSIS	1	YES	13	12	13	R4	X	B
95	MANIMEKALAI	55	F	COPD COR PULMONALE	2	NO	9	10	10	R4	X	A
96	AISHA BANU	52	F	BRONCHIAL ASTHMA	3	YES	14	13	13	R4	X	A
97	KARUNAIKATHIR	18	M	OPC POISONING	4	NO	10	12	13	R3	X	A
98	MUTHUKARUPPAN	72	M	CARCINOMA LUNG	3	NO	16	16	18	R1	Y	B
99	KALAISELVI	25	F	HERBICIDE POISONING	4	NO	6	7	7	R4	X	A
100	MATHIYAAS	45	M	PLEURAL EFFUSION	4	NO	8	8	7	R1	X	A